KU Leuven
Biomedical Sciences Group
Faculty of Kinesiology and Rehabilitation Sciences
Department of Rehabilitation Sciences

The role of proprioceptive postural control in the development and maintenance of low back pain:

A cross-sectional and a prospective study

Kurt CLAEYS

Promotor:

Prof. S. Brumange

Co-Promotor:

Prof. W. Dankaerts

Dissertation presented in partial fulfillment of the requirements for the degree of Doctor in Rehabilitation Sciences & Physiotherapy

November 2013

Jury

Promotor: Prof. Dr. Simon Brumagne

Co-Promotor: Prof. Dr. Wim Dankaerts

Chair: Prof. Dr. Luc Vanhees

Committee: Prof. Dr. Jaap van Dieën

Prof. Dr. Jaak Duysens

Prof. Dr. Martine Thomis

Prof. Dr. Filip Staes

Table of contents

Dankwoord

Een mensenleven kan soms enorme wendingen nemen. Voor- en tegenspoed zijn nooit veraf, voor je het weet overkom je van de ene op de andere dag enorme tegenslagen. Bepaalde zaken heb je in de hand, andere omstandigheden overvallen je zonder je er zelf wat vat op hebt. Zo is het ook met een professionele carriere. Je behaalt je diploma en tekent een bepaalde weg uit voor jezelf in het arbeidscircuit. Je interesses en verlangens kunnen wijzigen en je stuurt je traject wat bij. Oorspronkelijk kies je ervoor om voluit te gaan voor klinische praktijk met als hoofddoel de zorg voor patiënten. Gaandeweg komt de interesse voor het onderwijs en neem je kans om aan de slag te gaan in het hoger niet universitair onderwijs aan de Katholieke Hogeschool Brugge-Oostende (KHBO). In slechts 12 jaar zie je enkele cruciale omwentelingen in het Vlaamse onderwijslandschap gebeuren: het wegvallen van een driejarige graduaatsopleiding ten voordele van een vierjarige masteropleiding aan de hogeschool, het opstarten van een intensieve samenwerking met de universiteit en tot slot de volledige inkanteling van de opleiding Revalidatiewetenschappen en Kinesitherapie (RevaKi) in de Katholieke Universiteit van Leuven (KU Leuven). Onderweg krijg je de vraag of je met het oog op de toekomst niet aan een doctoraat zou denken. Je wimpelt oorspronkelijk af, maar komt al vlug tot het besef dat je een enorme kans hebt laten liggen. Je wordt door geluk gediend en de vraag komt terug. In een euforische bui reageer je voorzichtig: "het zou mij wel interesseren!". Vooraleer je het beseft krijg je een positief antwoord en mag je meteen contacten gaan leggen aan de KU Leuven. Meteen wil ik dan ook graag de vroegere KHBO danken voor de positieve houding om een personeelslid vrij te stellen van onderwijs ten voordele van het wetenschappelijk onderzoek. Het avontuur kon beginnen.

De man die je project stuurt en ook jouwzelf op het goede spoor zet, is je promotor. Simon, ik bewonder jouw enorme theoretische kennis, werklust en gedrevenheid. Je bent gepassioneerd door fundamentele wetenschap en onderliggende mechanismen. Je hebt me moeten wegwijs maken in jouw onderzoekslijn van lagerugpijn en proprioceptie. Mijn labo-ervaring was nihil en veel geduld was nodig van jouw kant om mij de tijd te geven om de nodige wetenschappelijke kennis te verwerken, temeer ik steeds maar deeltijds aan het project gewerkt heb. Feed-back via mail of tijdens een rechtstreeks gesprek laat nooit lang op zich wachten. Ongelofelijk bedankt voor de intensieve begeleiding tijdens mijn doctoraatsproject.

Daarnaast heeft dit project ook een co-promotor: Wim Dankaerts, bij de start van het project pas terug van een wetenschappelijk avontuur in Australië waar hij ook heel veel klinische expertise opdeed. Wim, jij waakte steeds over de link met de klinische praktijk. De tijdstippen waarop de mails van jou kwamen, suggereerden dat je nog steeds op Australische klok afgesteld stond. Ongelofelijk welke lange werkdagen jij kan kloppen. Ook jou dien ik verschrikkelijk te bedanken voor de goede afloop van dit project.

Naast twee promotoren kun je ook rekenen op een doctoraatsbegeleidingscommissie. Prof. Luc Vanhees, Prof. Jaak Duyssens, Prof. Martine Thomis en Prof. Filip staes hebben gedurende de afgelopen jaren hun specifieke wetenschappelijke expertise ten dienste gesteld aan dit project. Alle bijeenkomsten van de commissie gebeurden steeds in een zeer vriendschappelijke sfeer onder de deskundige coördinatie van de voorzitter. Steeds ging ik met zeer veel waardevolle tips terug naar huis. In de laatste fase kwamen daar ook nog de heel waardevolle suggesties bij van Prof. Jaap van Dieën, het externe jurylid. Ook aan jullie allen richt ik een welgemeende dankuwel.

Dit doctoraat kwam mede tot stand dankzij de vele uren testen in het labo. Die labotestings komen pas tot stand door de inzet van vele mensen: de proefpersonen die zich vrijwillig beschikbaar stellen, the thesis-studenten die moeten assisteren, collega-assisten die mee instaan voor de planning en uitvoering van de testen, ingenieurs die beschikbaar zijn als de apparatuur het laat afweten... Aan iedereen die hierin zijn rol gespeeld heeft, betuig ik mijn welgemeende dank. Ik zal bewust geen namen opsommen, want hoe langer de lijstjes die je maakt, hoe meer kans je iemand vergeet.

Daarnaast heb je de verschillende collega’s aan de hogeschool en aan de universiteit. Aan de Faculteit Bewegings- en Revalidatiewetenschappen van de KU Leuven kwam ik terecht in een team van jonge dynamische collega’s binnen de afdeling muskuloskeletale revalidatie. Samenwerken op de bureau, discussiëren over de verschillende doctoraatsonderwerpen, gaan eten in de alma, ... Deze hechte groep praktijkassistenten en onderzoekers slagen erin een teamgeest te vormen waardoor het lange werken geen opdracht is maar eerder een leuke ontspanning tussen de lange werkdagen in de praktijk. In één adem wil ik ook de collega's van de vroegere KHBO danken. Iedereen was steeds geïnteresseerd in de voortgang van het project. Sommige collega's waren steeds beschikbaar voor statistisch advies of als hulp bij de wiskundige software die mij niet eigen was. De extreme gedrevenheid van dit docententeam, gekoppeld aan die positieve teamspirit heeft mij niet alleen geholpen in mijn onderzoeksproject, maar heeft er ook voor gezorgd dat de opleiding RevaKi van de vroegere KHBO nog steeds bestaat in tegenstelling tot veel andere (West)vlaamse hogeschoolopleidingen die het academiseringsproces niet overleefd hebben.

Zo komen we terecht bij de nieuwe Brugse universitaire campus Kulab. Reeds enkele eeuwen onderneemt Brugge tevergeefse pogingen om een universiteit naar de stad te halen. Een laatste mislukte poging dateert uit de jaren 60: Kortrijk won het pleit en de Kulak was geboren. Maar door de niet aflatende inzet van één figuur is een eeuwenoude Brugse droom toch realiteit geworden. Dokter William De Groote heeft als voorzitter van de raad van bestuur van de KHBO en als ondervoorzitter van de associatie KU Leuven een cruciale rol gespeeld bij het tot stand komen van de Kulab. Bovendien had en heeft hij steeds een bijzondere belangstelling voor het reilen en zeilen binnen de opleiding RevaKi. Nog steeds stelt hij zijn kennis inzake orthopedie en traumatologie ter beschikking van de studenten als docent van de vakken orthopedische en traumatologische revalidatie. Zonder zijn niet aflatende inzet en die van het docententeam had West-Vlaanderen momenteel geen opleiding RevaKi meer.

Tot slot kom je terecht bij de mensen die jou het meest dierbaar zijn. De start van alles ligt bij je ouders, die je als 18-jarige de kans geven om een universitair diploma te behalen. Ook hier mag je gerust nog eens bij stilstaan, ook al ben je reeds 20 jaar afgestudeerd. Maar de mensen die het van meest nabij meegemaakt hebben, zullen bij de afwerking van dit boekje minimum even gelukkig zijn als ikzelf. Fran, ik hoef niet te herhalen dat het af en toe een harde combinatie was: een drukke praktijk, een man die twee tot soms drie maal per week in Leuven zit van 's morgens vroeg tot 's avonds heel laat, 2 kleine opgroeiende kinderen, ... Toch hebben we het samen klaargespeeld. Dit doctoraat is ook een beetje jouw doctoraat. Je stond er heel vaak alleen voor maar tegen de vermoeidheid in bleef jij ook volharden. Dankzij jou heb ik deze opdracht tot een goed einde kunnen brengen en kan ik mijn professionele activiteiten aan de Kulab, die ik bijzonder graag doe, verder zetten. Jarne en Myrthe, heel dikwijls hebben jullie gevraagd waarom papa alweer aan die PC moest zitten of waarom papa alweer naar Leuven moest? Talloze keren moesten jullie zonder papa op stap naar één of andere vrijetijdsactiviteit. Jullie vonden het niet leuk en wees gerust: ik ook niet. Aan deze periode zal nu wel een einde komen en de eerstvolgende reis die we allen samen maken, zal er eentje zijn zonder KU Leuven laptop. Dat is beloofd.

Kurt Claeys, Zerkegem, november 2013

Chapter 1

General introduction

1. Low back pain

Low back pain (LBP) and the reoccurrence of LBP is a major health problem in Western society with high social and economic consequences (Carragee et al. 2005). In a recent review it was demonstrated that LBP is most prevalent in people between 40 and 80 years old (Fig. 1) (Hoy et al., 2012). A large part of these persons is still working. As a consequence, LBP may affect a large part of the working population which may clarify the high economical cost for health insurance companies. However, clinicians in daily practice are convinced that the group of patients with LBP is becoming still younger. This opinion is confirmed in a review article of Jeffries et al. (2007). This study revealed that the incidence of LBP in the late adolescence approximates the incidence of the adult population. Moreover, LBP at adolescent age is mostly idiopathic and may higher the risk for future episodes of LBP (Jeffries et al., 2007).

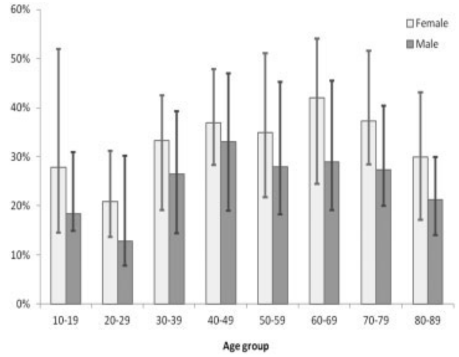


Fig. 1. Median prevalence of low back pain, with interquartile range, according to sex and midpoint of age group. Midpoint = (lower limit of age group + [upper limit of age group – lower limit of age group]/2) (Hoy et al., 2012)

Idiopathic LBP or non-specific LBP is defined as pain in the lumbar and/or gluteal region without structural anatomical abnormalities as there are disc abnormalities, inflammation, fracture, tumor, etc. (Dionne et al., 2008; Waddell, 1992). The absence of structural abnormalities indicates that other (functional) mechanisms may be associated in the development and reoccurrence of LBP. The biopsychosocial model of LBP was introduced at the beginning of the last decade of the last century (Waddell, 1992). Within this model pain may be caused or persist despite the absence of a nociceptive stimulus. An important functional mechanism responsible for LBP may be altered proprioceptive control. Decreased postural control in combination with altered proprioceptive steering (Brumagne et al., 2004; Brumagne et al., 2008a), reduced postural robustness (della Volpe et al., 2006), postural changes (Dankaerts et al., 2006; Mitchell et al., 2010) and changed postural strategy (Popa et al., 2007) are demonstrated as contributing factors in LBP and may confirm that a real anatomical nociceptive stimulus is not always present in people with LBP. Two remarks need to be considered concerning these studies: first, these studies are mostly cross-sectional and do not clarify the cause-effect relationship. Second, studies investigating proprioceptive postural control evaluate most the motor/postural control such as postural robustness, postural angles and muscle onsets after perturbations. However, they do not evaluate the sensory or proprioceptive input during the postural control tasks.

Last years, research into spinal pain is executed within a multifactorial framework. Not only biological, but also psychological factors and physical activity scores are investigated. There is a tendency to evaluate not only one type of variables but to investigate more types of variables simultaneously. As a result, besides the biological factors (e.g. proprioception, postural control) also psychological factors may be associated with the development and/or reoccurrence of LBP and must be evaluated in studies investigating proprioceptive postural control. Fear, stress, anxiety, somatization and kinesiophobia may play an important role in the development, maintenance and reoccurrence of LBP (Carragee et al., 2005; Janwantanakul et al., 2012; Mitchell et al., 2010; Terluin, 1998; Vlaeyen et al., 1995; Waddell et al., 1993).

However, research of the last two decades heavily focused on the psychological variables and the biological approach lost attention. A better understanding of the biological component of LBP in relation, and in addition to psychosocial factors, is important for a more rational approach to the management of LBP (Hancock et al., 2011). Research into biological underlying causes and mechanisms may be of priority interest for the research in LBP for the next years (Costa et al., 2013). In addition to cross-sectional research, prospective studies are necessary to have more insight in the underlying mechanisms of LBP.

2. Proprioceptive postural control

a. Proprioception

The term ‘proprioception’ was first described at the start of the 20th century (Sherrington CS, 1900). More recently it was defined as “the unconscious perception of movement and spatial orientation arising from stimuli within the body” (Stedman, 2002). Four components of proprioception can be distinguished (Proske, 2005; Proske and Gandevia, 2009): Kinaesthetic sense, sense of tension of force, sense of balance and sense of effort or heaviness.

Kinaesthetic sense, namely the sense of position and movement, is predominantly derived from the muscle spindles with few contribution of joint and skin receptors (Goodwin et al., 1972). The sense of tension is provided by the golgi tendon organs. These receptors operate more as detectors of the limits of the joint movements (Proske 2012). The sense of balance is provided by the vestibular system, the sense of effort or heaviness is believed to be a central phenomenon generated at the motor cortex (Gandevia et al. 1992; Gandevia 1996; Proske 2005; Proske and Gandevia 2009). Based on the studies of Goodwin et al. (1972) and Cordo et al. (1995) , it may be concluded that kinesthetic sense is the most important component in dynamic proprioception.

As a result kinaesthetic sense, predominantly derived from the muscle spindles, may have most attention in scientific research and in the clinical practice of rehabilitation (Brumagne et al., 2010). These sensory signals, together with the visual and vestibular inputs, are weighted by the central nervous system (CNS), appropriate to the postural activity and the postural condition. As a result, muscle contractions co-ordinated by the CNS may result in an optimal postural control strategy. This process, called sensory reweighting, is crucial to choose the optimal postural control strategy in order to achieve optimal postural control (Brumagne et al., 2004; Carver et al., 2006). Figure 2 shows the different sensory signals within the sensory reweighting process.

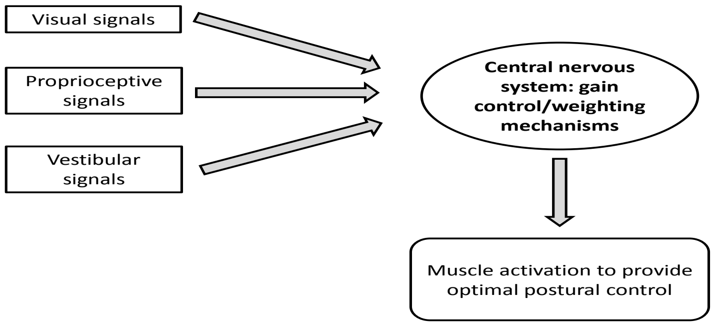


Fig. 2. The sensory signals important for the sensory reweighting process

People with LBP have been observed to have altered sensory reweighting. They may be more visual dependent to provide postural control compared to healthy subjects (della Volpe et al., 2006; Mientjes and Frank, 1999; Mok et al., 2004). More specific, they are unable to upweight other sensory signals (e.g. from the proprioceptive system) to achieve optimal control. As a result, this more visual dependent postural strategy is associated with a decreased postural robustness.

Furthermore, also within the proprioceptive system, a weighting of the afferent signals based on location is crucial. Healthy people are able to downweight proprioceptive signals from the calf muscles and upweight the multifidus muscles proprioceptive input when the postural task becomes more complex (e.g. standing on a foam vs. standing on stable support surface) (Brumagne et al., 2004). In contrast, elderly people and people with LBP are less capable of performing this proprioceptive reweighting, which results in a decreased postural robustness (Brumagne et al., 2004).

The term 'postural robustness' includes more than postural stability. Stability means that a system's behavior after perturbation is not significantly different from the original behavior (Reeves et al., 2007). In this context people have the possibility to return to their original positions after perturbation without falling. According to Reeves et al. (2007), the term stability is too limited concerning postural control: a system is either stable or it is not – there should be no index or level of stability. Commonly, the term stability is often confused with robustness. To clarify, it is more appropriate to say the system is more robust than more stable. Postural robustness is more complex and means that a system has the ability to maintain stable behavior despite small or large, sometimes unexpected perturbations (Reeves et al., 2007). Therefore, the system has to be able to change some parameters (e.g. stiffness or muscular co-contractions resulting in rigid single inverted pendulum strategy) without losing stability (Reeves et al., 2007). Consequently the system has the ability to rapidly and adequately react on expected perturbations and to coordinate more complex movement without losing stability. Therefore, feed-back from the muscle and joint proprioceptors with adequate programming of muscular contractions by the CNS is crucial in this process. Specifically, it means that people have the ability to explore between the safety margins of stability to provide optimal control. This process of exploring is very important in more complex postural positions, to provide optimal postural control without falling. However, most studies demonstrating proprioceptive deficits in people with LBP are based on cross-sectional analysis. Therefore, the cause-effect relation remains unclear. Are proprioceptive deficits caused by LBP and/or do proprioceptive deficits result in LBP?

Moreover, most studies evaluating postural control in people with LBP make an assumption of the proprioceptive impairments without a more direct measurement (della Volpe et al., 2006; Henry et al., 2006; Mok et al., 2004; Mok et al., 2007; Popa et al., 2007; Radebold et al., 2001). In the studies of Brumagne et al. (2004, 2008a, 2008b) the proprioceptive changes are evaluated by means of muscle vibration. Muscle vibration has been shown as a strong stimulus for muscle spindles (Goodwin et al., 1972; Roll and Vedel, 1982; Roll et al., 1989). Using muscle vibration on multifidus muscles and on calf muscles gives the opportunity to investigate which muscle spindle signals subjects are using predominantly to provide optimal postural control.

Earlier studies evaluating the proprioceptive system used repositioning tasks to evaluate the proprioceptive impairments in people with LBP. These studies showed conflicting results: some studies observed larger repositioning errors suggesting proprioceptive impairments in people with LBP (Brumagne et al., 2000; Descarreaux et al., 2005; Dolan and Green, 2006; Newcomer et al., 2000) while other studies could not demonstrate larger repositioning errors (Koumantakis et al., 2002; Silfies et al., 2007).

These results indicate some evidence that patients with LBP have reduced proprioceptive awareness, although other studies have questioned this (Koumantakis et al., 2002; Newcomer et al., 2000). The lack of significant differences in some studies may be due to heterogeneity within the LBP population (Koumantakis et al., 2002; Newcomer et al., 2000). There may be some patients with LBP with enhanced, rather than reduced, proprioceptive awareness (Hobbs et al., 2010; Mitchell et al., 2010), possibly reflecting a form of hypervigilance (Vlaeyen et al., 2002).

Again, most studies are cross-sectional and do not clarify a cause-effect relationship. Only the study of Silfies et al. (2007) was a prospective study in young sports men. Proprioception was evaluated by evaluating a repositioning error but this study could not identify a decreased repositioning accuracy as a risk factor in the development of LBP. In relation to studies evaluating repositioning errors as measure for the proprioceptive system two remarks need further attention. First, these studies did not evaluate proprioceptive impairments in combination with postural sway characteristics. As a result, it remains unclear if proprioceptive changes are associated with reduced postural robustness. Second, evaluating repositioning accuracy is more a memory task and thus an evaluation on a conscious level. In contrast, proprioceptive control during activities of daily life is more a subconscious process. As a result, a repositioning task may be less appropriate as an evaluation of the proprioceptive system.

b. Postural control

Optimal postural control is of crucial importance during daily activities. It gives people the possibility to keep equilibrium in standing or sitting posture. Postural adjustments assist to maintain upright standing or sitting posture by controlling the center of mass (COM) within the support base (Allum et al., 1998). Optimal sensory reweighting co-ordinated by the CNS may be crucial to choose the most appropriate postural control strategy according to the postural condition (Brumagne et al., 2004; Carver et al., 2006). To keep optimal postural control in the sagittal plane in upright standing position two models have been described: the inverted pendulum control model and the multisegmental control model (Allum et al., 1998; Horak and Nashner, 1986; Morasso and Schieppati, 1999; Runge et al., 1999).

The **inverted pendulum control model** is a postural control model in which the body pivots as a rigid segment around one joint, i.e. the ankle. Within this model, four strategies can be distinguished: an ankle strategy, a hip strategy, a suspensory strategy and a stepping strategy. An ankle strategy keeps upright posture by pivoting the body around the ankle joints (Horak and Nashner, 1986). This peripheral strategy may fail in complex postural tasks. As a result, a more efficient, proximal to distal inverted pendulum strategy is called a hip strategy. Here the postural corrections are primarily generated at the hip joints which may be more efficient and less energy demanding (Winter et al., 2003). Some people lower the COM by flexing the knees and the hips to maintain balance. This strategy is called a suspensory strategy (Nashner and McCollum G, 1985). A stepping strategy is used when people are not capable of maintaining balance. As a result they take a (forward) step to prevent falling (Horak and Nashner, 1986). The **multisegmental control model** is a postural control model where multiple corrections at different joints co-ordinated by the CNS keep upright standing balance (Kiemel et al., 2008; Morasso and Schieppati, 1999; Schieppati et al., 2002). The multisegmental postural control strategy is considered as the most optimal, with the least postural sway, during complex postural conditions. Moreover, within the multisegmental control model, an optimal control of the vertebrae is achieved by a fine-tuned segmental co-ordination of the different vertebrae by the deep segmental spinal muscles (Brumagne et al., 2010). Figure 3 shows an ankle-steered and a multisegemental steered postural control strategy in combination with the impact on the spine.

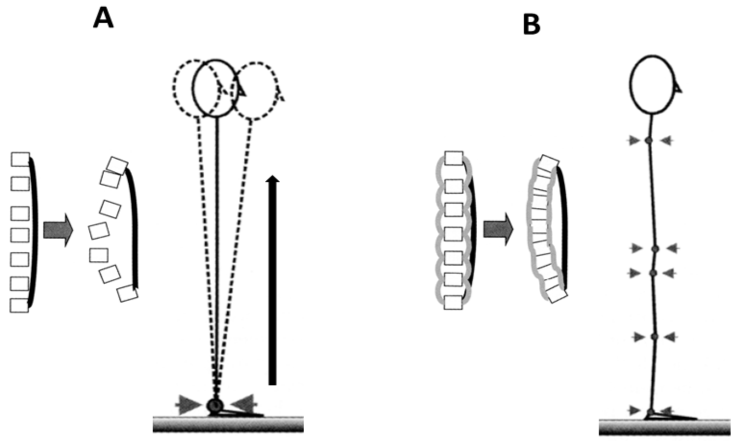


Fig. 3. An ankle-steered postural strategy (A) and a multisegmental steered postural strategy (B) in combination with their impact on the spine.

Altered postural control, more specifically decreased postural robustness, is frequently demonstrated in people with LBP. They may become more ankle-steered during standing with more forward body inclination (a more rigid postural strategy) (Brumagne et al., 2008a; Brumagne et al., 2008b). Increasing the complexity of the standing task, such as standing on an unstable or on a small support base, may evoke the postural control deficits in people with LBP (Brumagne et al., 2008b; Mientjes and Frank, 1999; Mok et al., 2004). Although decreased postural robustness is frequently observed in patients with LBP, the underlying mechanisms are still under discussion. Using an ankle strategy instead of a hip strategy may result in a decreased postural robustness (Mok et al., 2004). Moreover, individuals with LBP may have decreased anticipatory control at the lumbar spine and the pelvis during voluntary arm movements in standing postural tasks (Jacobs et al., 2009; Mok et al., 2007). Proprioceptive deficits are often mentioned as possible causing mechanisms in these studies, but were not directly evaluated. As a result, the contribution of proprioceptive impairments in the altered postural control in people with LBP during standing remains unclear.

People with LBP have been observed to have decreased postural robustness during sitting (Radebold et al., 2001). Delayed trunk muscle onset times are shown in this study. Moreover, altered proprioceptive signaling has been suggested to play a role, but this was not directly evaluated.

Furthermore, both in standing and sitting, a cause-effect relation still remains unclear by a lack of prospective studies. The few prospective studies that have been evaluating proprioceptive impairments as a risk factor in the development of LBP showed conflicting results. Delayed trunk muscle responses to sudden loading in sitting suggests that proprioceptive impairments may contribute to the development of LBP in young people (Cholewicki et al., 2005). This finding was not confirmed in the study of Silfies et al. (2007) where larger repositioning errors in sitting seemed not to be predictive for future LBP episodes. In contrast, more accurate spinal repositioning in sitting was identified as a risk factor in the development of LBP in young female nursing students (Mitchell et al., 2010). With regard to these conflicting results some shortcomings of these prospective studies need to be considered. First, only the sitting position was evaluated. Second, a more specific proprioceptive evaluation was not performed.

Both, standing and sitting are the two most frequently used daily postural positions. As a result, moving from sitting to standing is a very frequently performed movement task with a demonstrated average frequency of 60 performances a day (Dall and Kerr, 2010). If the performance of static postural tasks (e.g. standing, sitting) is impaired in people with LBP, consequently, the performance of dynamic tasks such as the sit-to-stance-to-sit task (STSTS) may be disturbed in people with LBP. Reduced lumbar and hip range of motion during this task has been seen in this population (Shum et al., 2005). Moreover, energy transfer from the pelvis to the lower limbs was decreased in people with LBP during the sit-to-stand (STS) transfer, which resulted in a greater energy demanding task for patients with LBP (Shum et al., 2009). These greater energy demands may initiate/exacerbate pain. These findings indicate that the performance of the STS movement in people with LBP is altered, but the underlying mechanism still remains unclear. Again, proprioceptive impairments may play a role in this changed strategy, but a direct evaluation was not performed in these studies.

To achieve optimal performance, optimal co-ordination of the COM during movements with large displacements (e.g. STS) is shown to be crucial to achieve optimal performance. This optimal co-ordination of the COM could be achieved by pelvic initiation of the movement such as during the sit-up (Cordo and Gurfinkel, 2004).

During the sit-up, this initial pelvic preparatory movement was demonstrated to facilitate a forward trunk motion by optimizing the moment arm of the psoas major muscle (Cordo et al., 2003). Also the STSTS movement requires substantial mass redistribution and shows impaired pelvic kinematics in people with LBP (Shum et al., 2005). The role of proprioceptive impairments as a possible underlying mechanism in the changed kinematics during the STSTS task remains unclear and requires further investigation.

Studies investigating postural control in people with LBP mainly focused on one single postural position (e.g. standing or sitting). As a result, information about the variability in this motor task performance remains unclear. However, variability is a fundamental property of biological systems and means that people have multiple options to perform one task based on adaptive strategies, rather than on rigid programs (Harbourne and Stergiou, 2009). It is reasonable that the optimal strategy in a stable standing condition is not appropriate in another postural position such as standing on an unstable support or sitting. Possibly, people with LBP have not only reduced postural control as already demonstrated but they may also show reduced variability in postural strategies between the different conditions. To have insight in the variability of postural control strategies, different postural positions need to be investigated. To have insight in the role of proprioception in the variability of postural control strategies, also the proprioceptive steering of the subjects must be evaluated.

Research on sitting and standing postures preceded the research of postural control. Posture is in the past defined as “the state of skeletal and muscular balance which protects the supporting structures of the body against injury or progressive deformity irrespective of the attitude in which these structures are working or resting; under such conditions the muscles will work efficiently and the most optimal positions are afforded for the thoracic and abdominal organs” (Kendall F.P. et al., 1993).

Kendall et al. (1993) described four postural types in standing based on sagittal X-rays: a ‘neutral’ posture, a ‘hyperlordotic’ posture (lumbar lordosis and thoracic kyphosis), a ‘flat back’ (flattened lumbar and thoracic curves) and a ‘sway back’ posture (backward displacement of the thoracic relative to the pelvis). A neutral posture was considered as the most optimal and less associated with all possible musculoskeletal problems such as LBP. More recently, these four postural types could also be distinguished using external markers in combination with sagittal photographs (Smith et al., 2008). Again, a neutral postural position (with ‘normal’ lumbar lordosis and thoracic kyphosis) was considered as the most optimal and providing a lower risk to develop LBP.

Several studies investigated the role of sagittal postural changes in LBP showing conflicting results. Changed sacral inclination angles are demonstrated to be associated with LBP (Evcik and Yucel, 2003; Tsuji et al., 2001). Adopting non-neutral sagittal postures may increase the risk for developing LBP (Black et al., 1996; Dunk et al., 2004; Dunk et al., 2005; Smith et al., 2008). In contrast, some studies could not demonstrate differences in usual standing or sitting posture in people with LBP (Mitchell et al., 2010). This prospective study only demonstrated that more posterior pelvic rotation during slump sitting increased the risk for developing LBP in nursing students. Two remarks should be mentioned according to these results: at first, the only risk factor was demonstrated in a targeted postural position (slump) and not in a usual postural position; second, only female persons were evaluated. These conflicting results emphasize the importance of more postural research in people with LBP and especially the role of posture as a possible causing factor of LBP.

Besides biological factors (e.g. proprioception and postural control), psychological factors can also influence pain processing in general and specifically in LBP (Moseley & Hodges 2006). In a prospective study Carragee et al. (2005) demonstrated that the development of serious LBP was strongly predicted by psychosocial factors in contrast to structural diseases (investigated by MRI and discography). High scores on the fear-avoidance beliefs questionnaire part physical activity (FABQ PA) were strongly predictive for future back pain episodes and the need for medical treatment. High scores on the FABQ PA questionnaire in people with LBP were also observed in cross-sectional studies (Davis et al., 2012). Furthermore, higher scores on the FABQ questionnaire in patient with LBP were associated with higher levels of disability and decreased quality of life. It is noticed that studies showing higher scores on psychosocial questionnaires mainly include people with moderate to high pain and disability scores. The current project aimed to have more insight in the role of proprioceptive postural control in the development/maintenance of LBP. Previous studies already demonstrated that psychosocial variables may play a role in the development/maintenance of LBP. As a result, these variables are also included, not as primary outcome measures but as secondary outcome measures (control variables).

3. Aims of this doctoral project

The **general objective** of this doctoral project is to generate a better insight in the proprioceptive postural control in young people with LBP versus healthy controls.

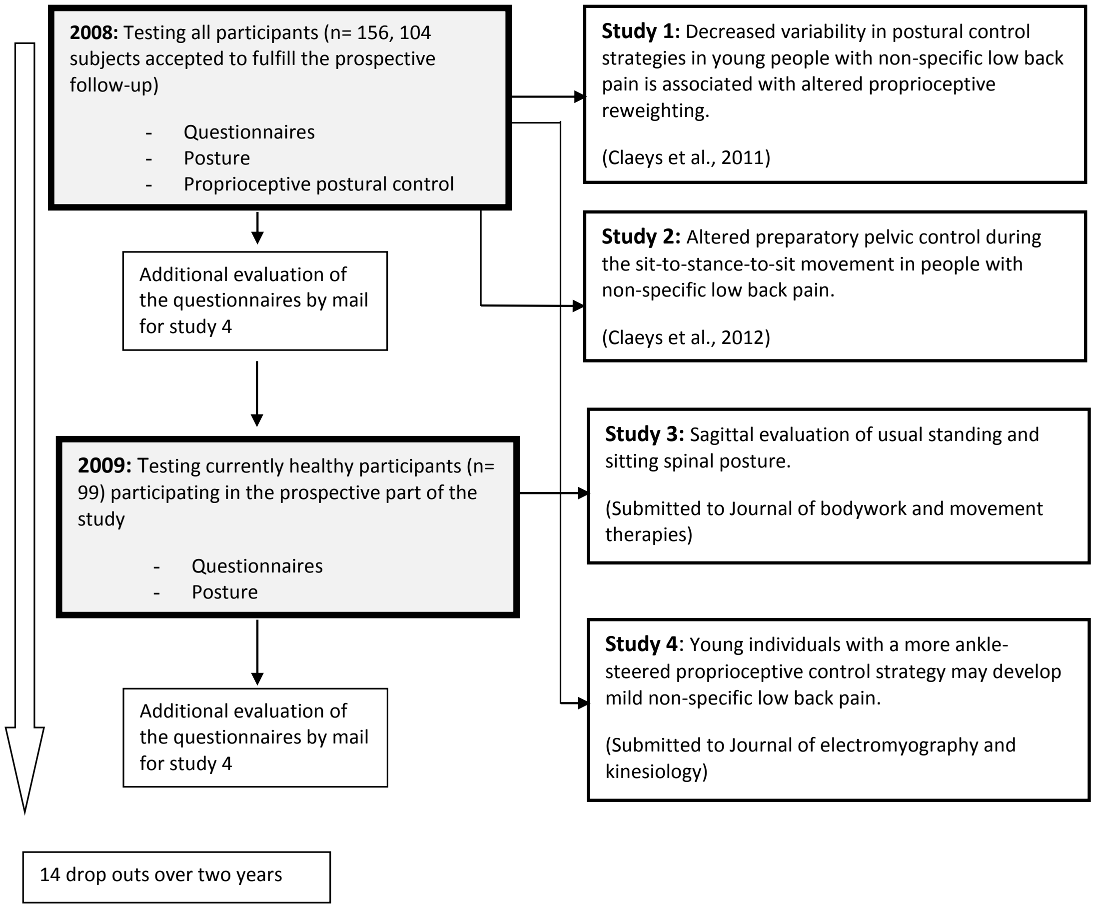
The first **specific aim** of this project is to investigate variability in proprioceptive postural control in young healthy persons and young persons with LBP.
Many studies confirmed the impaired proprioceptive postural control in people with LBP. However, most studies focused on one postural condition to investigate postural control. As a result, the variability in postural control strategies is not investigated. To have more insight in the variability in postural control strategies in healthy young people and in people with LBP three postural conditions were tested: standing on a stable support surface, standing on a foam pad and usual sitting. A force plate was used to identify the postural sway characteristics in both groups.
To have more insight in the proprioceptive steering of the subjects during the three postural tasks, muscle vibration was used. More sway during ankle muscle vibration indicates that people are adopting an ankle-steered postural strategy. However, more sway during multifidus muscle vibration indicates that people are adopting a more optimal multisegmental postural strategy for postural control. This first specific aim is addressed in Chapter 2 (Study 1).

The **second specific aim** is to investigate if proprioceptive impairments demonstrated during static postural tasks in people with LBP are associated with an altered performance of a more dynamic task. As a dynamic task, the STSTS task was chosen because of its clinical usefulness, it's good (inter- and intratester) reliability in healthy people as well as in people with LBP and its high frequency in daily life.
Study 2 aimed to evaluate the proprioceptive steering of people with LBP and healthy controls during standing, sitting and the performance of a dynamic task (the STSTS movement). This second specific aim is addressed in Chapter 3 (Study 2).

A **third specific aim** of this project is to investigate postural inter-correlations during usual standing and sitting. Markers on anatomical landmarks in combination with digital photographs in usual standing and sitting are used to calculate postural angles. The third specific aim is addressed in Chapter 4 (Study 3).

A **fourth specific aim** is to investigate if altered proprioceptive postural control could be identified as a risk factor for developing or sustaining LBP. Currently, the cause-effect relationship between LBP and altered proprioceptive postural control remains unclear due to the fact that most studies are cross-sectional in design.
Consequently, proprioceptive postural control characteristics were evaluated in a young population with and without LBP at baseline. The incidence of LBP was registered at baseline and during a two year follow-up period. At the end of the two years, intake variables of the subjects were analyzed to have more insight in their prognostic value to sustain or develop LBP. This fourth specific aim is addressed in Chapter 5 (Study 4).

4. Overview of the subjects in the different studies



5. References

1. Allum JH, Bloem BR, Carpenter MG, Hulliger M, Hadders-Algra M. Proprioceptive control of posture: a review of new concepts. Gait. Posture. 1998;8(3):214-242.
2. Black KM, McClure P, Polansky M. The influence of different sitting positions on cervical and lumbar posture. Spine 1996;21(1):65-70.
3. Brumagne, S. Het sensorimotorische systeem: controle van houding en beweging in de lumbosacrale wervelkolom., in P Vaes, Kwakkel G., Smits-Engelsman B.C.M., and AP Verhagen eds., Jaarboek Kinesitherapie 2002; Houten (NL), Bohn Stafleu Van Loghum, p. 108-143.
4. Brumagne S, Cordo P, Lysens R, Verschueren S, Swinnen S. The role of paraspinal muscle spindles in lumbosacral position sense in individuals with and without low back pain. Spine 2000;25(8):989-994.
5. Brumagne S, Cordo P, Verschueren S. Proprioceptive weighting changes in persons with low back pain and elderly persons during upright standing. Neurosci. Lett. 2004;366(1):63-66.
6. Brumagne, S, Dolan P, and Pickar JG. Chapter 19: What is the relation between proprioception and low back pain? In: Hodges, PW, Cholewicki J, Van Dieën J. (Eds), Spinal Control: The Rehabilitation of Back Pain - State of the Art and Science. 2013;Elsevier Churchill Livingstone, Edinburgh, p. 219-230.ISBN:978-0-7020-4356-7.
7. Brumagne, S, Janssens L, Claeys K, and Pijnenburg M.Chapter 12: Altered variability in proprioceptive postural strategy in people with recurrent low back pain and healthy individuals. In:Hodges, PW, Cholewicki J., Van Dieën J. (Eds),Spinal Control: The Rehabilitation of Back Pain - State of the Art and Science. 2013;Elsevier Churchil Livingstone, Edinburgh, p. 135-144.ISBN:978-0-7020-4356-7.
8. Brumagne S, Janssens L, Janssens E, Goddyn L. Altered postural control in anticipation of postural instability in persons with recurrent low back pain. Gait. Posture. 2008a;28(4):657-662.

9. Brumagne S, Janssens L, Knapen S, Claeys K, Suuden-Johanson E. Persons with recurrent low back pain exhibit a rigid postural control strategy. Eur. Spine J. 2008b;17(9):1177-1184.
10. Carragee EJ, Alamin TF, Miller JL, Carragee JM. Discographic, MRI and psychosocial determinants of low back pain disability and remission: a prospective study in subjects with benign persistent back pain. Spine J. 2005;5(1):24-35.
11. Carver S, Kiemel T, Jeka JJ. Modeling the dynamics of sensory reweighting. Biol. Cybern. 2006;95(2):123-134.
12. Cholewicki J, Silfies SP, Shah RA, Greene HS, Reeves NP, Alvi K, Goldberg B. Delayed trunk muscle reflex responses increase the risk of low back injuries. Spine 2005;30(23):2614-2620.
13. Cordo PJ, and Gurfinkel VS. Motor coordination can be fully understood only by studying complex movements. Prog. Brain Res. 2004;14329-38.
14. Cordo PJ, Gurfinkel VS, Smith TC, Hodges PW, Verschueren SM, Brumagne S. The sit-up: complex kinematics and muscle activity in voluntary axial movement. J. Electromyogr. Kinesiol. 2003;13(3):239-252.
15. Cordo PJ, Gurfinkel VS, Bevan L, Kerr K. Proprioceptive consequences of tendon vibration during movement. J. Neurophysiol 1995;74(4):1675-1688.
16. Costa LC, Koes BW, Pransky G, Borkan J, Maher CG, Smeets RJ. Primary care research priorities in low back pain: an update. Spine (Phila Pa 1976.) 2013;38(2):148-156.
17. Dall PM, and Kerr A. Frequency of the sit to stand task: An observational study of free-living adults. Appl. Ergon. 2010;41(1):58-61.
18. Dankaerts W, O'Sullivan P, Burnett A, Straker L. Differences in sitting postures are associated with nonspecific chronic low back pain disorders when patients are subclassified. Spine 2006;31(6):698-704.
19. Davis DS, Mancinelli CA, Petronis JJ, Bensenhaver C, McClintic T, Nelson G. Variables Associated With Level of Disability in Working Individuals With Non-Acute Low Back Pain: A Cross-Sectional Investigation. J. Orthop. Sports Phys. Ther. 2013;43(2):97-10

20. della Volpe R, Popa T, Ginanneschi F, Spidalieri R, Mazzocchio R, Rossi A. Changes in coordination of postural control during dynamic stance in chronic low back pain patients. Gait. Posture. 2006;24(3):349-355.
21. Descarreaux M, Blouin JS, Teasdale N. Repositioning accuracy and movement parameters in low back pain subjects and healthy control subjects. Eur. Spine J. 2005;14(2):185-191.
22. Dionne CE et al. A consensus approach toward the standardization of back pain definitions for use in prevalence studies. Spine 2008;33(1):95-103.
23. Dolan KJ, and Green A. Lumbar spine reposition sense: the effect of a 'slouched' posture. Man. Ther. 2006;11(3):202-207.
24. Dunk NM, Chung YY, Compton DS, Callaghan JP. The reliability of quantifying upright standing postures as a baseline diagnostic clinical tool. J. Manipulative Physiol. Ther. 2004;27(2):91-96.
25. Dunk NM, Lalonde J, Callaghan JP. Implications for the use of postural analysis as a clinical diagnostic tool: reliability of quantifying upright standing spinal postures from photographic images. J. Manipulative Physiol Ther. 2005;28(6):386-392.
26. Evcik D, and Yucel A. Lumbar lordosis in acute and chronic low back pain patients. Rheumatol. Int. 2003;23(4):163-165.
27. Gandevia SC. Kinesthesia, roles for afferent signals and motor command. In: Rowell LB, Shephard JT (Eds.). Handbook of physiology, section 12 exercise: regulation and integration of musltiple systems. Oxford University Press, New York, pp. 128-172.
28. Gandevia SC, McCloskey DI, Burke D. Kinaesthetic signals and muscle contraction. Tends. Neurosci. 1992;15:62-65.
29. Goodwin GM, McCloskey DI, Matthews PB. Proprioceptive illusions induced by muscle vibration: contribution by muscle spindles to perception? Science 1972;175(28):1382-1384.
30. Hancock MJ, Maher CG, Laslett M, Hay E, Koes B. Discussion paper: what happened to the 'bio' in the bio-psycho-social model of low back pain? Eur. Spine J. 2011;20(12):2105-2110.

31. Harbourne RT, and Stergiou N. Movement variability and the use of nonlinear tools: principles to guide physical therapist practice. Phys. Ther. 2009;89(3):267-282.
32. Henry SM, Hitt JR, Jones SL, Bunn JY. Decreased limits of stability in response to postural perturbations in subjects with low back pain. Clin. Biomech. 2006;21(9):881-892.
33. Hobbs AJ, Adams RD, Shirley D, Hillier TM. Comparison of lumbar proprioception as measured in unrestrained standing in individuals with disc replacement, with low back pain, and without low back pain. J. Orthop. Sports Phys. Ther. 2010;40(7):439-446.
34. Horak FB, and Nashner LM. Central programming of postural movements: adaptation to altered support-surface configurations. J. Neurophysiol. 1986;55(6):1369-1381.
35. Hoy D, Bain C, Williams G, March L, Brooks P, Blyth F, Woolf A, Vos T, Buchbinder R. A systematic review of the global prevalence of low back pain. Arthritis Rheum. 2012;64(6):2028-2037.
36. Jacobs JV, Henry SM, Nagle KJ. People with chronic low back pain exhibit decreased variability in the timing of their anticipatory postural adjustments. Behav. Neurosci. 2009;123(2):455-458.
37. Janwantanakul P, Sitthipornvorakul E, Paksaichol A. Risk factors for the onset of nonspecific low back pain in office workers: a systematic review of prospective cohort studies. J. Manipulative Physiol. Ther. 2012;35(7):568-577.
38. Jeffries LJ, Milanese SF, Grimmer-Somers KA. Epidemiology of adolescent spinal pain: a systematic overview of the research literature. Spine 2007;32(23):2630-2637.
39. Kendall FP, McCreary EK, Provance PG. Muscles: Testing and Function, With Posutre and Pain. 4th ed. 1993. Baltimore, Williams & Wilkins.
40. Kiemel T, Elahi AJ, Jeka JJ. Identification of the plant for upright stance in humans: multiple movement patterns from a single neural strategy. J. Neurophysiol. 2008;100(6):3394-3406.

41. Koumantakis GA, Winstanley J, Oldham JA. Thoracolumbar proprioception in individuals with and without low back pain: intratester reliability, clinical applicability, and validity. J. Orthop. Sports Phys. Ther. 2002;32(7):327-335.
42. Mientjes MI, and Frank JS. Balance in chronic low back pain patients compared to healthy people under various conditions in upright standing. Clin. Biomech. 1999;14(10):710-716.
43. Mitchell T, O'sullivan PB, Burnett A, Straker L, Smith A, Thornton J, Rudd CJ. Identification of modifiable personal factors that predict new-onset low back pain: a prospective study of female nursing students. Clin. J. Pain 2010;26(4):275-283.
44. Mok NW, Brauer SG, Hodges PW. Hip strategy for balance control in quiet standing is reduced in people with low back pain. Spine (Phila Pa 1976.) 2004;29(6):E107-E112.
45. Mok NW, Brauer SG, Hodges PW. Failure to use movement in postural strategies leads to increased spinal displacement in low back pain. Spine 2007;32(19):E537-E543.
46. Morasso PG, and Schieppati M. Can muscle stiffness alone stabilize upright standing? J. Neurophysiol. 1999;82(3):1622-1626.
47. Nashner LM, and McCollum G. The organization of human postural movements: a formal basis and experimental synthesis. Behav. Brain Res. 1985;8135-172.
48. Newcomer KL, Laskowski ER, Yu B, Johnson JC, An KN. Differences in repositioning error among patients with low back pain compared with control subjects. Spine 2000;25(19):2488-2493.
49. Popa T, Bonifazi M, Della VR, Rossi A, Mazzocchio R. Adaptive changes in postural strategy selection in chronic low back pain. Exp. Brain Res. 2007;177(3):411-418.
50. Proske U. What is the role of muscle receptors in proprioception? Muscle Nerve 2005;31(6):780-787.
51. Proske U, and Gandevia SC. The kinaesthetic senses. J. Physiol 2009;587(Pt 17):4139-4146.

52. Proske U, and Gandevia SC. The proprioceptive senses: their roles in signaling body shape, body position and movement and muscle force. Physiol Rev. 2012;92(4):1651-97
53. Radebold A, Cholewicki J, Polzhofer GK, Greene HS. Impaired postural control of the lumbar spine is associated with delayed muscle response times in patients with chronic idiopathic low back pain. Spine 2001;26(7):724-730.
54. Reeves NP, Narendra KS, Cholewicki J. Spine stability: the six blind men and the elephant. Clin. Biomech. 2007;22(3):266-274.
55. Roll JP, and Vedel JP. Kinaesthetic role of muscle afferents in man, studied by tendon vibration and microneurography. Exp. Brain Res. 1982;47(2):177-190.
56. Roll JP, Vedel JP, Ribot E. Alteration of proprioceptive messages induced by tendon vibration in man: a microneurographic study. Exp. Brain Res. 1989;76(1):213-222.
57. Runge CF, Shupert CL, Horak FB, Zajac FE. Ankle and hip postural strategies defined by joint torques. Gait. Posture. 1999;10(2):161-170.
58. Schieppati M, Giordano A, Nardone A. Variability in a dynamic postural task attests ample flexibility in balance control mechanisms. Exp. Brain Res. 2002;144(2):200-210.
59. Sherrington CS, 1900, The muscular sense. Textbook of physiology., Edinburgh, Scafer EA (Ed.), p. 1002-1025.
60. Shum GL, Crosbie J, Lee RY. Effect of low back pain on the kinematics and joint coordination of the lumbar spine and hip during sit-to-stand and stand-to-sit. Spine 2005;30(17):1998-2004.
61. Shum GL, Crosbie J, Lee RY. Energy Transfer Across the Lumbosacral and Lower-Extremity Joints in Patients With Low Back Pain During Sit-to-Stand. Arch. Phys. Med. Rehabil. 2009;90:127-35.
62. Silfies SP, Cholewicki J, Reeves NP, Greene HS. Lumbar position sense and the risk of low back injuries in college athletes: a prospective cohort study. BMC. Musculoskelet. Disord. 2007;8129.
63. Smith A, O'Sullivan P, Straker L. Classification of sagittal thoraco-lumbo-pelvic alignment of the adolescent spine in standing and its relationship to low back pain. Spine 2008;33(19):2101-2107.

64. Stedman. Stedman's Medical Dictionary Houhton Mifflin Company; 2002.
65. Terluin,B. De Vierdimensionele Klachtenlijst (4DKL) in de huisarspraktijk - psychodiagnostisch gedreedschap. 1998;3318-24..
66. Tsuji T, Matsuyama Y, Sato K, Hasegawa Y, Yimin Y, Iwata H. Epidemiology of low back pain in the elderly: correlation with lumbar lordosis. J. Orthop. Sci. 2001;6(4):307-311.
67. Vlaeyen JW, de Jong JR, Onghena P, Kerckhoffs-Hanssen M, Kole-Snijders AM. Can pain-related fear be reduced? The application of cognitive-behavioural exposure in vivo. Pain Res. Manag. 2002;7(3):144-153.
68. Vlaeyen JW, Kole-Snijders AM, Boeren RG, van EH. Fear of movement/(re)injury in chronic low back pain and its relation to behavioral performance. Pain 1995;62(3):363-372.
69. Waddell G. Biopsychosocial analysis of low back pain. Baillieres Clin. Rheumatol. 1992;6(3):523-557.
70. Waddell G, Newton M, Henderson I, Somerville D, Main CJ. A Fear-Avoidance Beliefs Questionnaire (FABQ) and the role of fear-avoidance beliefs in chronic low back pain and disability. Pain 1993;52(2):157-168.
71. Winter DA, Patla AE, Ishac M, Gage WH. Motor mechanisms of balance during quiet standing. J. Electromyogr. Kinesiol. 2003;13(1):49-56.

Chapter 2

Decreased variability in postural control strategies in young people with non-specific low back pain is associated with altered proprioceptive reweighting

***Adapted from European Journal of Applied Physiology*. 2011;111:115–123.**

Kurt Claeys[1,2], Simon Brumagne[1], Wim Dankaerts[1,3], Henri Kiers[4], Lotte Janssens[1]

1. Research group Musculoskeletal Rehabilitation, KU Leuven Department of Rehabilitation Sciences, , University of Leuven, Leuven, Belgium
2. Department Health Care, Catholic University College of Bruges-Ostend, Bruges
3. Department of Health Care, University College Limburg, AUHL-PHL, REVAL - Rehabilitation and Health Care Research Center, Hasselt, Belgium
4. Department Lifestyle and Health, Hogeschool Utrecht, University of Applied Sciences

Abstract

Optimal postural control is an essential capacity in daily life and can be highly variable. The purpose of this study was to investigate if young people have the ability to choose the optimal postural control strategy according to the postural condition and to investigate if non-specific low back pain (LBP) influences the variability in proprioceptive postural control strategies. Young individuals with LBP (n= 106) and healthy controls (n= 50) were tested on a force plate in different postural conditions (i.e., sitting, stable support standing and unstable support standing). The role of proprioception in postural control was directly examined by means of muscle vibration on triceps surae and lumbar multifidus muscles. Root mean square (RMS) and mean displacements of the center of pressure (COP) were recorded during the different trials. To appraise the proprioceptive postural control strategy, the relative proprioceptive weighting (RPW, ratio of ankle muscles proprioceptive inputs versus back muscles proprioceptive inputs) was calculated. Postural robustness was significantly less in individuals with LBP during the more complex postural conditions ($p < 0.05$). Significantly higher RPW-values were observed in the LBP group in all postural conditions ($p < 0.05$), suggesting less ability to rely on back muscle proprioceptive inputs for postural control. Therefore, healthy controls seem to have the ability to choose a more optimal postural control strategy according to the postural condition. In contrast, young people with LBP showed a reduced capacity to switch to a more multi-segmental postural control strategy during complex postural conditions, which leads to decreased postural robustness.

Key words: Postural control, multi-segmental strategy, ankle strategy, sensory reweighting, variability, low back pain.

1. Introduction

Optimal postural control is an essential capacity in daily activities and can be highly variable. For example, the maintenance of quiet stance can be performed through adjustments at the ankles, knees, hips and spine (Allum et al., 1998). When postural conditions change, the central nervous system (CNS) must identify and selectively focus on the most reliable sensory inputs to provide optimal control. Inputs from the vestibular, the visual and the proprioceptive system are weighted by the CNS. As a result of this weighting, muscle forces can be produced to control the center of mass efficiently to maintain a good equilibrium (Brumagne et al., 2004; Carver et al., 2006). Previous studies described different models and strategies to maintain optimal postural control in the sagittal plane during standing (Horak and Nashner, 1986; Runge et al., 1999). Within the 'inverted pendulum' postural control model, where the body pivots as a rigid segment around one joint, two strategies can be distinguished. An ankle strategy restores the equilibrium by moving the body primarily around the ankle joints (Horak and Nashner, 1986). While this strategy could be sufficient in simple postural conditions such as standing on a flat surface, in more complex postural tasks it might fail. To achieve optimal stability in more difficult postural conditions, according to the inverted pendulum model, the resulted motion to maintain balance is primarily generated at the trunk and the hips (i.e., hip strategy) (Horak and Nashner, 1986). In contrast, according to the 'multi-segmental' postural control model, postural control is achieved by multiple corrections at different joints coordinated by the CNS and not only by corrections at one joint (Morasso and Schieppati, 1999; Schieppati et al., 2002; Kiemel et al., 2008).

One factor that could disturb the optimal multi-segmental postural control is non-specific low back pain (LBP). Individuals with LBP have been observed to have decreased postural robustness during standing, particularly when the standing task becomes more difficult such as standing on an unstable support surface (Mientjes and Frank, 1999; Mok et al., 2004; della Volpe et al., 2006). Furthermore, individuals with LBP have shown poorer postural control during sitting (Radebold et al., 2001; O'Sullivan et al., 2006; Dankaerts et al., 2006). Both during standing (Mok et al., 2004) and during sitting (Radebold et al., 2001; Van Daele et al., 2009) impaired proprioception has been suggested as a possible mechanism causing the impaired postural control. Although a specific assessment of the impaired proprioception was not performed in these studies.

Several studies already evaluated proprioceptive changes in people with LBP by determining the lumbosacral position sense. Larger repositioning errors in people with LBP suggest proprioceptive impairments (Newcomer et al., 2000; Brumagne et al., 2000; Descarreaux et al., 2005; Dolan and Green, 2006), while other studies could not demonstrate larger repositioning errors and associated impaired proprioception (Koumantakis et al., 2002; Silfies et al., 2007). However, these studies did not evaluate repositioning errors in combination with postural sway characteristics. So, it remains unclear if proprioceptive impairments are associated with reduced postural robustness. Moreover, an evaluation of postural sway characteristics in combination with muscle vibration evaluates the subconscious proprioceptive control, while repositioning tasks are more an evaluation on a conscious level (e.g., rely more on memory) and therefore less representative of normal proprioceptive control.

Within the proprioceptive system, reweighting of sensory signals has already been demonstrated in both healthy controls and in individuals with LBP (Brumagne et al., 2004). Another study investigating two postural standing conditions (standing on stable and unstable support) in a larger test population already suggested decreased variability of postural control strategy in people with LBP (Brumagne et al., 2008a). These studies only evaluated standing postural conditions, so the role of proprioceptive reweighting as a characteristic of variability in postural control strategies (during standing as well as in sitting conditions) was not evaluated specifically. Variability, as a fundamental property of biological systems, means that the person has multiple options to perform one task based on adaptive strategies, rather than on rigid programs (Harbourne and Stergiou, 2009). To have more insight in the variability of postural control strategies and its changes in people with LBP, it is recommended to investigate this under a variety of different postural conditions (e.g., standing on a stable and on an unstable surface as well as sitting).

A decrease in variability of anticipatory adjustments (APAs) during postural control has been observed in experimental induced acute LBP and in recurrent LBP (Moseley and Hodges, 2006; Jacobs et al., 2009). Pain related beliefs have been suggested as a possible mechanism for this decreased variability in postural strategy in both studies (Moseley and Hodges, 2006; Jacobs et al., 2009), but the role of proprioception was not evaluated in these studies. Furthermore, subjects were only tested during one condition (standing), so it remains unclear if the persons had the variability to choose the optimal strategy upon different postural conditions. To get more insight in the selection variability of postural control strategies upon the condition and the possible role of impaired proprioception, investigating the specific role of proprioception during diverse postural conditions is essential.

Therefore, this study had two aims. *The first aim* of this study was to investigate if healthy people show variability in their proprioceptive postural control strategy to ensure postural robustness during increased postural complexity. To investigate this first aim, different postural conditions (standing on a stable and unstable surface and sitting) were chosen. Furthermore, muscle vibration, known as a strong stimulus for muscles spindles (Roll and Vedel, 1982), was used to more specifically appraise the role of proprioception. *The second aim* was to investigate if age-matched people with LBP show a similar variability in postural strategy. It was hypothesized that healthy persons regulate their proprioceptive postural control strategy depending on the postural demands and that individuals with LBP demonstrate less variability by selecting the same ankle-steered strategy independent of the postural condition.

2. Materials and methods

Subjects

One hundred fifty-six students (47 men, 109 women) volunteered in this study. All subjects were included or excluded in the study by an experienced musculoskeletal physical therapist. Exclusion criteria were a history of vestibular disorders, neurological or respiratory disease, previous spinal surgery, structural spinal problems, acute radiculopathy, serious neck problems and recent musculoskeletal problems (< 6months). All subjects had to fill out four questionnaires: a physical activity questionnaire (Baecke et al., 1982), the Oswestry disability index (ODI-2) (Fairbank and Pynsent, 2000), the fear avoidance beliefs questionnaire (FABQ) (Waddell et al., 1993) and the Tampa scale of Kinesiophobia (Vlaeyen et al., 1995). In addition, they had to score the pain at the moment of testing on a numerical rating scale (NRS). Subjects were included in the LBP group if they reported a NRS > 0 and if they scored ODI-2 > 6 at the moment of the test. The healthy controls did not report any pain (NRS= 0) and had an ODI-2 score of 0. Characteristics of all subjects are presented in Table 1.

All subjects gave their written informed consent and all test procedures were approved by the Medical Research Ethics Committee of KU Leuven with respect to the declaration of Helsinki (Ethical Principles for Medical Research Involving Human Subjects).

Table 1. Characteristics of the test population

	Healthy Controls N = 50		**Persons with LBP N = 106**		**P**
	Mean	**SD**	**Mean**	**SD**	
Male	17		25		
Female	33		81		
Age (years)	19.6	1.6	18.5	0.5	NS
Height (cm)	171.4	7.9	170.9	9.1	NS
Weight (kg)	64.3	8.9	63.2	8.5	NS
BMI	21.89	2.3	21.63	2.4	NS
PAI (5-15)	8.5	1.4	7.9	1.9	NS
NRS (0-10)	0	0	2	2.2	S
ODI (0-100)	0	0	8.8	2.0	S
FABQPA (0-24)	4.4	5.8	7.9	5.3	NS
FABQW (0-42)	3.0	6.0	4.6	7.6	NS
TSK (17-68)	31.1	5.5	33.1	4.9	NS

The values are means with standard deviations, BMI = Body Mass Index, PAI = Physical Activity Index (work index + sport index + leisure-time index, max. score: 5 + 5 + 5 = 15), NRS = pain at the moment of the test scored on a numeric rating scale (0-10), ODI = score on the 0swestry Disability Index (min. 0 – max 100), FABQPA = Fear Avoidance Beliefs Questionnaire Physical Activity (min. 0 – max. 24) , FABQW = Fear Avoidance Beliefs Questionnaire Work (min. 0 – max. 42), TSK = Tampa Scale for Kinesiophobia (min. 17 – max. 68), NS = not significant, LBP = Non Specific Low Back Pain, P < 0.05 means significant difference.

Movement analysis

Postural sway characteristics were measured using a six-channel force plate (Bertec Corporation, OH, U.S.A.). Force plate data were sampled at 500 Hz using a Micro 1401 data-acquisition system and Spike2 software (Cambridge Electronic Design, U.K.) and low pass filtered with a cutoff frequency of 5 Hz. To evaluate trunk position in space, two piezo-resistive accelerometers (ICSensors, U.K.), also connected with the data-acquisition system, were placed on the spinous processes of thoracic (T1) and sacral (S2) vertebra in upright posture.

Muscle vibration

In six trials, the role of proprioception in postural control was directly examined by means of muscle vibration, known as a powerful stimulus of Ia afferents (Roll and Vedel, 1982). Therefore, two muscle vibrators (self-manufactured with Maxon motors, Switzerland) were used. Vibration was applied bilaterally to triceps surae muscles or to lumbar multifidus muscles, respectively. These muscles were selected, based on previous studies to represent the muscles used in an ankle-steered strategy or a multi-segmental strategy, respectively (Brumagne et al., 2008b). Muscle vibration was initiated 15 s after the start of the trial for duration of 15 s. Activation and deactivation of the vibrators were manually controlled. The frequency of the vibration was set at 60 Hz and the amplitude was approximately 0.5 mm. These characteristics of vibration were chosen to induce maximal illusory joint movement and were demonstrated to induce a significant muscle lengthening illusion in healthy individuals (Roll and Vedel, 1982; Cordo et al., 2005). When the CNS is using the signals of the vibrated muscles for postural control, larger directional sways are expected. When triceps surae muscles are vibrated in a healthy subject during standing, a postural sway in backward direction is expected; when lumbar multifidus muscles are vibrated during standing, a healthy subject is expected to show a postural sway in a forward direction. The effect of lumbar multifidus muscle vibration will be different depending on the reference frame the CNS is using (Gurfinkel et al., 1995; Paulus and Brumagne, 2008). During standing, the sacrum-pelvis will be considered as the ‘mobile’ body part compared to the ‘stationary’ trunk. So the resulting illusion of lumbar multifidus muscle lengthening during vibration corresponds with a posterior pelvic tilting and thus a posterior COP displacement. Therefore, the subject will compensate this illusion with a forward COP displacement. During sitting, however, the trunk will be considered as the ‘mobile’ body part compared to the ‘stationary’ sacrum-pelvis which is connected to the stool. Consequently, the illusion during lumbar multifidus muscle vibration corresponds with a trunk flexion and thus an anterior COP displacement. Hence, the subject will compensate this kinesthetic illusion with a backward COP displacement during sitting.

Test procedure

To appraise postural stability and proprioceptive postural control in quiet stance, two test conditions were used: (1) an upright standing condition on a stable support surface and (2) an upright standing condition on an unstable support surface ("foam"), respectively. To appraise proprioceptive postural control in sitting, subjects sat on a stable stool with the feet stable. The sitting condition was chosen to evaluate the possibility to switch to a more appropriate postural control strategy when the postural condition changes (e.g., from standing to sitting). Table 2 gives an overview of all postures and the different trials.

Table 2. The experimental trials to evaluate postural stability and proprioceptive postural control

Posture: Quiet standing	
Condition 1: stable support surface	
Trial 1	Quiet standing
Trial 2	Quiet standing, ballistic shoulder flexion to 90° at 30 s.
Trial 3	Quiet standing, bilateral triceps surae vibration
Trial 4	Quiet standing, bilateral lumbar multifidus muscle vibration
Condition 2: unstable support surface (foam)	
Trial 5	Quiet standing
Trial 6	Quiet standing, ballistic shoulder flexion to 90° at 30 s.
Trial 7	Quiet standing, bilateral triceps surae vibration
Trial 8	Quiet standing, bilateral lumbar multifidus muscle vibration
Posture: stable sitting	
Trial 9	Sitting
Trial 10	Sitting, bilateral triceps surae vibration
Trial 11	Sitting, bilateral lumbar multifidus muscle vibration

In all standing conditions, subjects had to stand barefoot on the force plate (Trials 1, 2, 3, 4) or on the "foam" (Trials 5, 6, 7, 8) with the arms loosely hanging along the body, both heels 10 cm separated and the forefeet in a free splayed out position. Feet position is standardized in all trials using a transparency sheet to mark off both feet. The "foam" condition is used to create a postural condition in which ankle proprioceptive signals are less reliable and therefore the CNS should rely more on other proprioceptive signals to control posture (Ivanenko et al., 1999).

In all sitting trials (9, 10, and 11) subjects sat on a stable stool with height adjusted to create a rectangle between the greater trochanter – lateral femoral condyle line and the lateral femoral condyle – lateral malleolus line, respectively. Feet position was standardized using the same transparency sheet from the standing trials. Subjects were asked to adopt a usual sitting posture with the arms loosely hanging along the body.

In all trials vision was occluded and subjects were asked to remain as immobile, but relaxed as possible in upright standing or usual sitting posture, respectively. Before the actual muscle vibration trial, subjects were familiarized with the vibration stimulus by experiencing a short bout of vibration (1-2 seconds) in order to minimize startle effects.

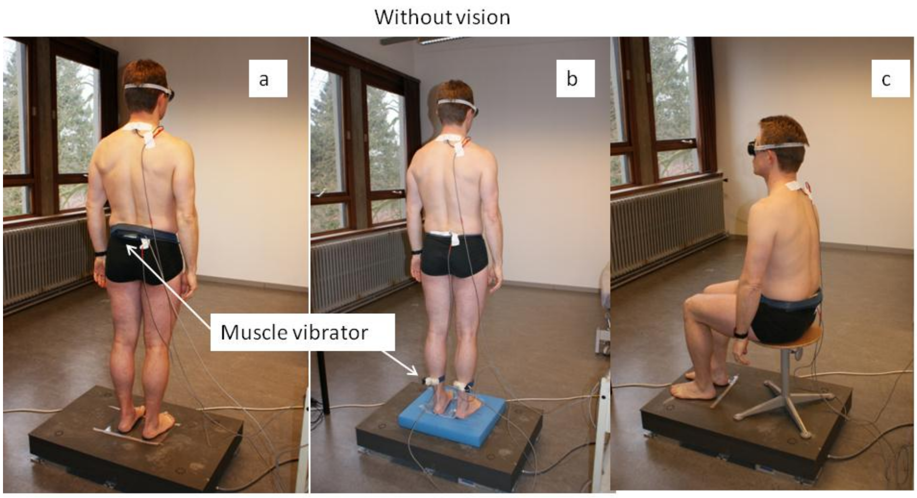


Fig. 1. Experimental set-up: a. Standing on a stable support; b. Standing on an unstable support ('foam') with application of muscle vibration on triceps surae muscles; c. Usual sitting on an adjustable stool.

Data reduction and statistical analysis

Postural sway characteristics from the force plate readings were collected and calculated using spike2 (CED, Cambridge U.K.) and Microsoft Excel software, for all trials of both groups. Displacements of the center of pressure (COP) in anterior-posterior direction were estimated from the raw force plate data using the equation:

$$\mathrm{COP} = \frac{Mx}{\mathrm{Fz}}$$

Further data reduction was performed by calculating the root mean square (RMS) values of the COP displacements for the stability trials (1, 2, 5, 6,) and the mean values for the muscle vibration trials in order to appraise the directional effect of muscle vibration on COP displacement. The COP displacements in the muscle vibration trials were analyzed over two epochs: the 15 s preceding and the 15 s during muscle vibration. Positive values correspond to forward COP displacement, negative values correspond to backward displacement. Furthermore, proprioceptive control strategy or relative proprioceptive weighting (RPW) was appraised using the equation:

$$RPW\frac{TS}{\mathrm{LM}} = \frac{(abs\ TS)}{(abs\ TS + abs\ LM)}$$

where abs TS is the absolute value of the mean COP displacement during triceps surae muscle vibration and abs LM is the absolute value of the mean COP displacement during lumbar multifidus muscle vibration. A score equal to 1 corresponds to 100% reliance on triceps surae muscle afference. A score equal to 0 corresponds to 100% reliance on lumbar multifidus muscle afference.

Differences in RMS and mean values of COP displacement between the conditions, between the trials, and between the LBP and healthy group were compared, based on repeated measures analysis of variance (ANOVA/MANOVA). Where a significant main and interaction effect was found post hoc tests (Tukey's unequal N HSD) were performed to further analyze the detailed effects. All data are presented as means ± standard deviations (SD). The level of statistical significance was set at $p \leq 0.05$. The statistical analysis was performed with Statistica 9 (Statsoft, OK, USA).

3. Results

Postural robustness

In the usual standing trial on a stable surface the subjects with LBP showed significantly less sway compared to the healthy group (Trial 1; p < 0.0001). However, the individuals with LBP showed significantly larger sways when standing on a foam support (Trial 5; p < 0.0001) and while performing a ballistic arm movement on an unstable support surface (Trial 6; p < 0.0001). Figure 2 illustrates the results of the stability trials.

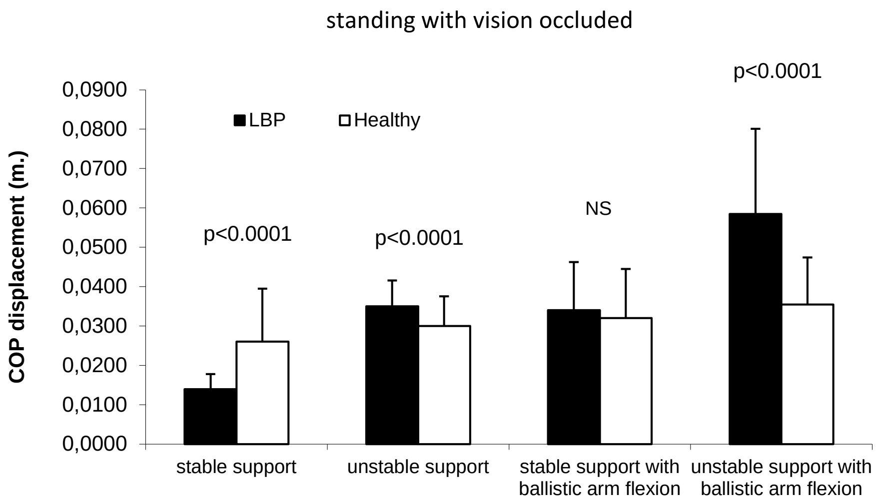


Fig. 2. RMS-values of the center of pressure (COP) displacement for the ‘baseline’ and ‘ballistic arm movement’ trials in the stable support surface and foam condition (LBP= non-specific low back pain, NS= not significant).

Proprioceptive postural control strategy expressed as relative proprioceptive weighting (RPW)

When standing on a stable support surface, individuals with LBP performed significantly more backward sway during triceps surae muscle vibration (Trial 3; $p < 0.0001$) compared to the healthy group. Muscle vibration on lumbar multifidus muscles showed no significant differences between the two groups (Trial 4; $p > 0.05$). In the foam conditions, however, larger forward sways were observed by the healthy control group compared to the individuals with LBP when vibration is applied on the lumbar multifidus muscles (Trial 8; $p < 0.0001$). In addition, during sitting, significantly larger sways were recorded when vibration was applied on lumbar multifidus muscle in the healthy group compared to the people with LBP (Trial 11; healthy: 0.0010 m. vs. LBP: 0.0000 m.; $p < 0.0001$). Figure 3 illustrates the results of the vibration trials during standing on a firm support surface and standing on foam; figure 4 illustrates the results of the vibration trials during sitting.

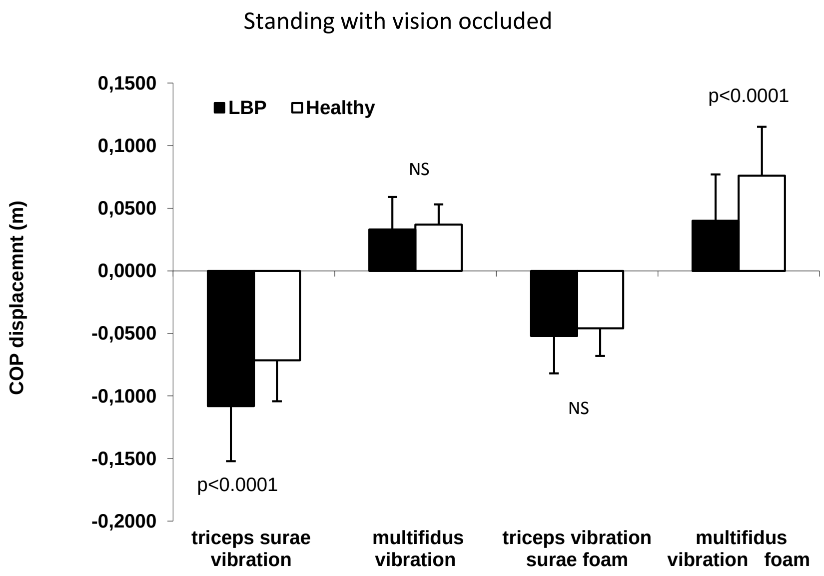


Fig. 3. Mean displacements of the center of pressure (COP) during the vibration trials for both groups when standing on a stable support surface and on ‘foam’, respectively (LBP= non-specific low back pain, NS= not significant).

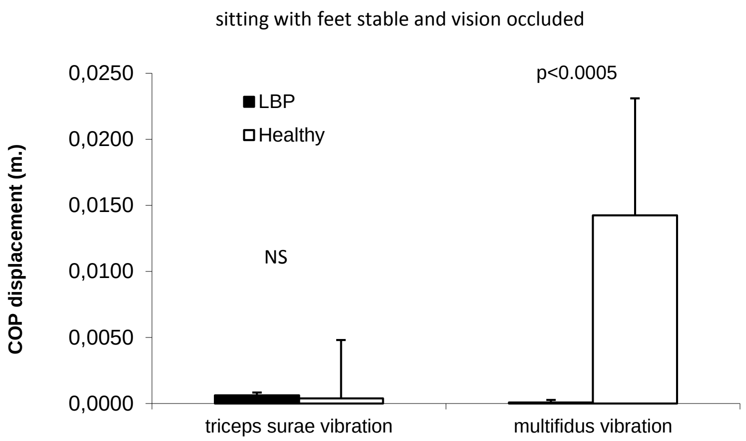


Fig. 4. Mean displacements of the center of pressure (COP) during the vibration trials for both groups during sitting (LBP= non-specific low back pain, NS= not significant).

Subjects with LBP showed significantly higher RPW values compared to healthy individuals both during standing on a stable support surface and on an unstable support surface ($p < 0.0001$). Also, during sitting, significantly higher RPW values were demonstrated in the LBP group ($p < 0.0001$). Figure 5 displays the RPW values for both groups during the different postural conditions.

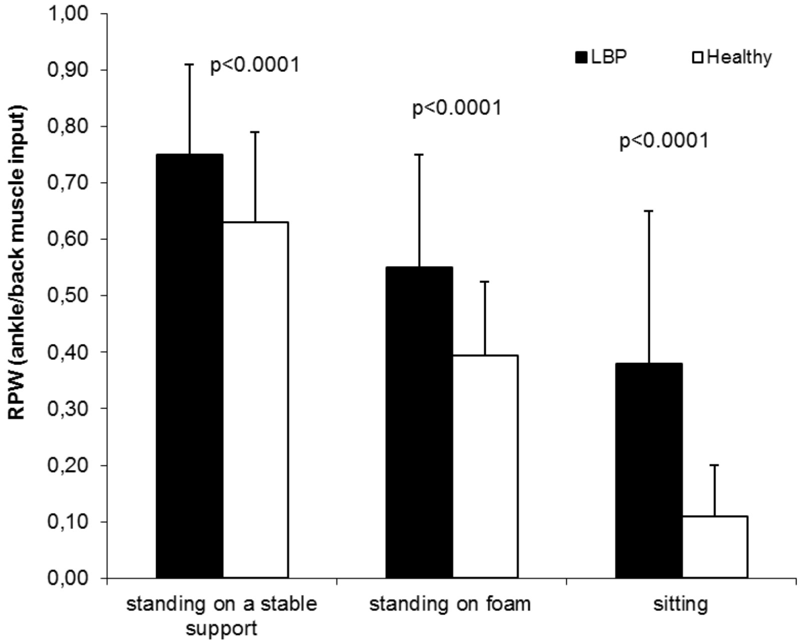


Fig. 5. Relative Proprioceptive Weighting (RPW) values of both groups during standing on stable support surface and on foam and during sitting. Higher RPW values mean more reliance on proprioceptive inputs of ankle muscles (LBP= non-specific low back pain).

4. Discussion

The main finding of this study is that young people with LBP (compared to healthy controls) show a stronger ankle-steered proprioceptive postural strategy during standing on a stable support surface and in a condition were this strategy is less appropriate (i.e., standing on an unstable support surface). This may lead to decreased postural robustness during standing. Furthermore, in a condition were lumbar proprioceptive afference is expected to be crucial (sitting), persons with LBP are not able to rely on this afference to control posture.

Postural robustness

Results of this study demonstrate greater anterior-posterior (AP) sways of the COP when standing on an unstable support surface and when performing a ballistic arm movement on an unstable support surface in people with LBP (Fig 2). These findings are in agreement with the results of previous research, where larger anterior-posterior (AP) sways were found when the postural task became more difficult and vision was occluded (Mientjes and Frank, 1999; Mok et al., 2004; Henry et al., 2006; Popa et al., 2007). Moreover, our findings are in agreement with earlier results where the integration of somatosensory signals during postural control of a young healthy population was tested (Isableu and Vuillerme, 2006). They found that the less the subjects swayed in a stable condition, the more they swayed in an unstable support surface (foam) condition. Our results show that in an easy postural condition (e.g., standing on a firm surface) the LBP group demonstrated less sway than the healthy group where the healthy group was more robust in all other (more complex) postural conditions. These findings might indicate that the adopted postural strategy of persons with LBP is effective for easy postural conditions; however, this postural strategy seems to fail in more complex postural conditions leading to decreased postural robustness.

Proprioceptive postural control strategy or relative proprioceptive weighting (RPW)

In the present study, we used muscle vibration, known as a powerful stimulus of muscle spindles, to evaluate the role of proprioception in postural control directly (Roll and Vedel, 1982; Cordo et al., 2005). It is demonstrated that (1) people with LBP demonstrate larger backward sways during triceps surae muscle vibration when standing on a stable support compared to the control group and that (2) healthy people demonstrate significantly more forward sway during multifidus muscle vibration when standing on a foam support. These findings illustrate clearly that proprioceptive differences are an influencing factor in this strategy selection. Previous studies already demonstrated more reliance on ankle signals during stable standing conditions in people with LBP (Brumagne et al., 2004; Brumagne et al., 2008a).

The higher RPW-values in the group with LBP compared to healthy controls when standing on a stable or an unstable support and during sitting, indicate that the LBP group relies less on back muscle proprioceptive inputs independent the postural condition. This reduced multi-segmental strategy seems to be adequate in stable support conditions, but leads to decreased postural robustness in unstable support conditions. Lumbosacral proprioceptive deficits were suggested as a possible reason why people with LBP could not switch to a more appropriate postural control strategy upon the condition (Mok et al., 2004), but a specific evaluation of the proprioceptive system did not occur.

These results are in accordance with previous studies showing that people with LBP used a more ankle-steered strategy to maintain upright position while healthy controls predominantly use a hip strategy when tested on a translational platform (Henry et al., 2006) or on a rotational platform (della Volpe et al., 2006). In contrast, Isableu et al. (2006) demonstrated that some healthy people use an ankle strategy in all conditions. This strategy results in less sway in the stable condition because people are not exploring for stability in this condition, which is in accordance with the people with LBP in the current study. However, exploring for stability may result in larger safety margins which may be a benefit in the more complex postural positions were larger safety margins are required to prevent falling. Healthy people in the current study and some people in the study of Isableu et al. (2006) have the capacity to explore which results in larger sways in the easiest condition but in less sway in the most complex postural position. The capacity to explore between safety margins to provide optimal stability is called postural robustness (Reeves et al., 2007). These findings also suggest that there might be differences in strategy selection in young healthy people, based on different central or peripheral proprioceptive processing. It may be possible that these people are at higher risk to get LBP in the future, due to impaired proprioception of the lumbosacral area. It is hypothesized that these proprioceptive changes may cause less fine-tuned control of the spine during postural control which may increase the risk to induce more mechanical stress on the spinal column causing (recurrent) LBP (Hodges and Moseley, 2003). Prospective studies investigating the role of reduced variability caused by altered proprioceptive inputs in postural control strategies are needed to clarify the role of reduced variability as a causing factor for LBP.

Furthermore, during sitting people with LBP also show higher RPW-values compared to the healthy controls. This suggests that they use less afferent signals from the back muscles in a condition where this signals are expected to play a predominant role in postural control. These results underscore the hypothesis that in LBP lumbosacral proprioceptive impairment is associated with decreased postural control variability. This finding is in accordance with earlier results where greater anterior-posterior (AP) sways of the COP during unstable sitting without vision are shown in people with LBP (Radebold et al., 2001).

The underlying mechanism causing the altered proprioceptive steering in people with LBP remains still unclear. Morphological, histochemical and neurophysiological changes are already shown in the lumbar multifidus in people with LBP (MacDonald et al. 2006). However, the relation between these changes and altered proprioceptive steering remains unclear. A possible mechanism underlying the decreased reliance on back muscle proprioception in patients with LBP might be a different muscle spindle density in the paraspinal muscles. Muscle spindles tend to concentrate mainly where oxidative muscle fibers predominate, often in the deeper and central portions of muscles (Kokkorogiannis, 2004). Individuals with LBP have been observed to have more fatigable muscle fibers due to decreased oxidative capacity (Mannion et al., 1997) and therefore might have a decreased density of muscle spindles in their back muscles. Consequently, lumbar multifidus vibration might cause a smaller effect. This hypothesis warrant further investigation.

Variability in postural control strategies

Few studies demonstrated a reduced variability of anticipatory control in young healthy people and age-matched persons with LBP (Moseley & Hodges 2006; Jacobs et al. 2009). In these studies, fear of movement and pain related beliefs were suggested as influencing factors. Brumagne et al. (2008b) demonstrated that only healthy persons show lower RPW-values during standing on an unstable support surface compared to a firm surface condition, whereas people with LBP showed similar RPW-values in both postural conditions. These earlier findings are in contrast with our findings demonstrating that healthy controls have lower RPW-values in all postural conditions. Thus, our results demonstrated that both young people with LBP as well as healthy controls have the ability to make a proprioceptive switch, but this capacity to switch is observed to be reduced in people with LBP. When subjects are about five years older, this proprioceptive variability may be further reduced (Brumagne et al., 2008b). These findings suggest that age may be an important factor in the capacity of varying postural control strategies and the associated sensory reweighting variability. It might be possible that at a certain age, young people with LBP move from adaptive ‘switchable’ postural control strategies to more rigid postural control strategies based on less variability in somatosensory reweighting. Prospective studies in different age groups are necessary to further explore this hypothesis.

In addition, in this study, fear may be likely ruled out as a causing factor for the reduction in postural strategy variability as there were no significant differences in the scores on FABQ en TSK questionnaires between the LBP and the control group. Moreover, the people with LBP had a low mean pain score of 2/10 at the time of testing, so pain is probably not the predominant factor responsible for the reduction in proprioceptive postural strategy variability.

Limitations and future directions

Some limitations of our study warrant discussion. First, a very young population with minimal pain and disability scores was investigated. Therefore, the results of this study cannot be generalized to the average LBP patient population. Moreover, a sub-classification based on pain aggravating movements and postures was not made. It could be possible that the people with LBP are still robust in some pain-free postures, but less robust in the pain aggravating postures. Another limitation of this study is that only rather static postural conditions (sitting and standing) were tested. Investigating the role of proprioceptive adaptations during more dynamic tasks (e.g., sit-to-stand, lifting a weight, forward bending) could give more insights in the role of proprioception in postural control.

Muscle vibration, used to appraise the proprioceptive steering, could be influenced by skin thickness. However, in the current study there were no significant differences in height and weight between both groups. Moreover, body mass indexes of both groups were fairly low (Table 1). Therefore, differences in COP displacements between the two groups during multifidus muscle vibration are unlikely attributed to lumbar skin thickness.

While more optimal investigation of proprioceptive changes may optimize treatment of persons with LBP, it remains difficult in clinical practice to evaluate these changes without the use of precise and accurate instruments as in a laboratory setting. Therefore, based on the laboratory results, a functional clinical test battery should be developed and its concurrent validity should be assessed before wider integration into clinical practice.

In addition, it might be fruitful to pay more attention on increasing the variability in postural strategies in rehabilitation of LBP. A large amount of multi-segmental postural correction possibilities depending on the postural task should be included in exercise programs. Performing postural control exercises on different surfaces (e.g., stable, unstable) and in different postural conditions (e.g., standing, sitting) might optimize this proprioceptive variability.

5. Conclusion

Young healthy people have the ability to choose the optimal multi-segmental postural control strategy according to the postural condition. In contrast, young people with mild LBP exhibit a reduced variability in proprioceptive postural control strategies due to a decreased proprioceptive reweighting capacity. This loss of variability in strategy selection is associated with a decreased postural robustness. Prospective studies are needed to further clarify the relation between reduced variability of postural control strategies and the development, reoccurrences or maintenance of LBP.

6. References

1. Allum JH, Bloem BR, Carpenter MG, Hulliger M, and Hadders-Algra M. Proprioceptive control of posture: a review of new concepts. Gait.Posture. 1998; 8(3):214-242.
2. Baecke JA, Burema J, and Frijters JE. A short questionnaire for the measurement of habitual physical activity in epidemiological studies. Am.J.Clin.Nutr. 1982;36(5):936-942.
3. Brumagne S, Cordo P, Lysens R, Verschueren S, and Swinnen S. The role of paraspinal muscle spindles in lumbosacral position sense in individuals with and without low back pain. Spine 2000;25(8):989-994.
4. Brumagne S, Cordo P, and Verschueren S. Proprioceptive weighting changes in persons with low back pain and elderly persons during upright standing: Neurosci.Lett. 2004;366(1):63-66.
5. Brumagne S, Janssens L, Janssens E, and Goddyn L. Altered postural control in anticipation of postural instability in persons with recurrent low back pain. Gait.Posture. 2008a;28(4):657-62.
6. Brumagne S, Janssens L, Knapen S, Claeys K, and Suuden-Johanson E. Persons with recurrent low back pain exhibit a rigid postural control strategy: Eur.Spine J.2008b;17(9):1177–1184.
7. Carver S, Kiemel T, and Jeka JJ. Modeling the dynamics of sensory reweighting. Biol.Cybern. 2006;95(2):123-134.
8. Cordo PJ, Gurfinkel VS, Brumagne S, and Flores-Vieira C. Effect of slow, small movement on the vibration-evoked kinesthetic illusion. Exp.Brain Res.2005;167(3):324-334.
9. Dankaerts W, O'Sullivan P, Burnett A, and Straker L. Altered patterns of superficial trunk muscle activation during sitting in nonspecific chronic low back pain patients: importance of subclassification. Spine 2006;31(17):2017-2023.
10. della Volpe R, Popa T, Ginanneschi F, Spidalieri R, Mazzocchio R, and Rossi A. Changes in coordination of postural control during dynamic stance in chronic low back pain patients: Gait.Posture.2006;24(3):349-355.

11. Descarreaux M, Blouin JS, and Teasdale N. Repositioning accuracy and movement parameters in low back pain subjects and healthy control subjects: Eur.Spine J. 2005;14(2):185-191.
12. Dolan KJ, and Green A. Lumbar spine reposition sense: the effect of a 'slouched' posture. Man.Ther.2006;11(3):202-207.
13. Fairbank JC, and Pynsent PB. The Oswestry Disability Index 5. Spine2000;25(22):2940-2952.
14. Gurfinkel VS, Ivanenko Y, Levik Y, and Babakova IA. Kinesthetic reference for human orthograde posture. Neuroscience1995; 68(1):229-243.
15. Harbourne RT, and Stergiou N. Movement variability and the use of nonlinear tools: principles to guide physical therapist practice. Phys.Ther.2009;89(3):267-282.
16. Henry SM, Hitt JR, Jones SL, and Bunn JY. Decreased limits of stability in response to postural perturbations in subjects with low back pain. Clin.Biomech.2006;21(9):881-892.
17. Hodges PW, and Moseley GL. Pain and motor control of the lumbopelvic region: effect and possible mechanisms. J.Electromyogr. Kinesiol. 2003;13(4):361-370.
18. Horak FB, and Nashner L. Central programming of postural movements: adaptation to altered support-surface configurations. J. Neurophysiol. 1986;55(6):1369-1381.
19. Isableu B, and Vuillerme N. Differential integration of kinaesthetic signals to postural control. Exp.Brain Res. 2006;174(4):763-768.
20. Ivanenko YP, Talis VL, and Kazennikov OV. Support stability influences postural responses to muscle vibration in humans: Eur.J.Neurosci. 1999;11(2):647-654.
21. Jacobs JV, Henry SM, and Nagle KJ. People with chronic low back pain exhibit decreased variability in the timing of their anticipatory postural adjustments: Behav. Neurosci. 2009;23(2):455-458.
22. Kiemel T, Elahi AJ, and Jeka JJ. Identification of the plant for upright stance in humans: multiple movement patterns from a single neural strategy. J. Neurophysiol. 2008;100(6):3394-3406.
23. Kokkorogiannis T. Somatic and intramuscular distribution of muscle spindles and their relation to muscular angiotypes. J.Theor. Biol. 2004;229(2):263-280.

24. Koumantakis GA, Winstanley J, and Oldham JA. Thoracolumbar proprioception in individuals with and without low back pain: intratester reliability, clinical applicability, and validity. J. Orthop. Sports Phys.Ther.2002;32(7):327-335.
25. Mannion AF, Weber BR, Dvorak J, Grob D, and Muntener M. Fibre type characteristics of the lumbar paraspinal muscles in normal healthy subjects and in patients with low back pain. J. Orthop. Res. 1997;15(6):881-887.
26. Mientjes MI, and Frank JS. Balance in chronic low back pain patients compared to healthy people under various conditions in upright standing. Clin. Biomech. 1999;14(10):710-716.
27. Mok NW, Brauer SG, and Hodges PW. Hip strategy for balance control in quiet standing is reduced in people with low back pain: Spine 2004;29(6):E107-E112.
28. Morasso PG, and Schieppati M. Can muscle stiffness alone stabilize upright standing? J. Neurophysiol. 1999;82(3):1622-1626.
29. Moseley GL, and Hodges PW. Reduced variability of postural strategy prevents normalization of motor changes induced by back pain: a risk factor for chronic trouble? Behav. Neurosci. 2006;120(2):474-476.
30. Newcomer KL, Laskowski ER, Yu B, Johnson JC, and An KN. Differences in repositioning error among patients with low back pain compared with control subjects. Spine2000;25(19):2488-2493.
31. O'Sullivan PB, Mitchell T, Bulich P, Waller R, and Holte J.The relationship between posture and back muscle endurance in industrial workers with flexion-related low back pain. Man.Ther.2006;11(4):264-271.
32. Paulus I, and Brumagne S. Altered interpretation of neck proprioceptive signals in persons with subclinical recurrent neck pain. J. Rehabil. Med. 2008;40(6):426-432.
33. Popa TM, Della Bonifazi VR, Rossi A, and Mazzocchio R. Adaptive changes in postural strategy selection in chronic low back pain. Exp.Brain Res. 2007;177(3): 411-418.
34. Radebold A, Cholewicki J, Polzhofer GK, and Greene HS. Impaired postural control of the lumbar spine is associated with delayed muscle response times in patients with chronic idiopathic low back pain. Spine 2001;26(7):724-730.
35. Reeves NP, Narendra KS, Cholewicki J. Spine stability: the six blind men and the elephant. Clin. Biomech. 2007;22(3):266-274.

36. Roll JP, and Vedel JP. Kinaesthetic role of muscle afferents in man, studied by tendon vibration and microneurography. Exp. Brain Res.1982;47(2):177-190.
37. Runge CF, Shupert CL, Horak FB, and Zajac FE. Ankle and hip postural strategies defined by joint torques. Gait. Posture.1999;10(2):161-170.
38. Schieppati M, Giordano A, and Nardone A. Variability in a dynamic postural task attests ample flexibility in balance control mechanisms. Exp. Brain Res. 2002;144(2):200-210.
39. Silfies SP, Cholewicki JN, Reeves P, and Greene HS. Lumbar position sense and the risk of low back injuries in college athletes: a prospective cohort study. BMC. Musculoskelet. Disord. 2007;8:129.
40. Van Daele U, Hagman F, Truijen S, Vorlat P, Van GB, and Vaes P. Differences in balance strategies between nonspecific chronic low back pain patients and healthy control subjects during unstable sitting. Spine2009;34(11):1233-1238.
41. Vlaeyen JW, Kole-Snijders AM, Boeren RG, and van EH. Fear of movement/(re)injury in chronic low back pain and its relation to behavioral performance. Pain1995;62(3):363-372.
42. Waddell G, Newton M, Henderson I, Somerville D, and Main CJ. A Fear-Avoidance Beliefs Questionnaire (FABQ) and the role of fear-avoidance beliefs in chronic low back pain and disability. Pain1993;52(2):157-168.

Chapter 3

Altered preparatory pelvic control during the sit-to-stance-to-sit movement in people with non-specific low back pain

***Adapted from Journal of Electromyography and Kinesiology*. 2012; 22:821-828.**

Kurt Claeys[1,2], Wim Dankaerts[1], Lotte Janssens[1], Simon Brumagne[1]

1. Research group Musculoskeletal Rehabilitation, KU Leuven Department of Rehabilitation Sciences,, University of Leuven, Leuven, Belgium
2. Department Health Care, Catholic University College of Bruges-Ostend, Bruges, Belgium

Abstract

People with non-specific low back pain (LBP) show hampered performance of dynamic tasks such as sit-to-stance-to-sit movement. However, the underlying mechanisms remain obscure. Therefore, the aim of this study was to assess if proprioceptive impairments influence the performance of the sit-to-stance-to-sit movement.

First, the proprioceptive steering of 20 healthy subjects and 106 persons with mild LBP was identified during standing using muscle vibration. Second, five sit-to-stance-to-sit repetitions on a stable support and on foam were performed as fast as possible. Total duration, phase duration, center of pressure (COP) displacement, pelvic and thoracic kinematics were analyzed.

People with LBP used less lumbar proprioceptive afference for postural control compared to healthy people ($P < 0.0001$) and needed more time to perform the five repetitions in both postural conditions ($P < 0.05$). These time differences were determined in the stance and sit phases (transition phases), but not in the focal movement phases. Moreover, later onsets of anterior pelvic rotation initiation were recorded to start both movement sequences ($P < 0.05$) and to move from sit-to-stance on foam ($P < 0.05$).

Decreased use of lumbar proprioceptive afference in people with LBP seemed to have a negative influence on the sit-to-stance-to-sit performance and more specifically on the transition phases which demand more control (i.e. sit and stance). Furthermore, slower onsets to initiate the pelvis rotation to move from sit-to-stance illustrate a decrease in pelvic preparatory movement in the LBP group.

1. Introduction

Optimal postural control is an essential quality in daily life. Inputs from the visual, vestibular and somatosensory system are weighted by the central nervous system (CNS). As a result, the body reacts efficiently with the most optimal muscle forces to keep the center of mass (COM) within the support base and thus provide adequate postural robustness (Carver et al., 2006).

This optimal control is demonstrated to be affected in patients with non-specific low back pain (LBP) (Mientjes and Frank, 1999). Decreased postural robustness is shown both in standing (Mok et al., 2004; Henry et al., 2006; Mok et al., 2007) as in sitting postural conditions in this population (Radebold et al., 2001). The lumbosacral region seems to play a crucial role within these impairments. Moreover, reduced proprioceptive afference (Brumagne et al., 2008b), altered proprioceptive reweighting in combination with decreased variability of postural strategies (Claeys et al., 2011), failure to use a hip-steered strategy (Mok et al., 2004), delayed onsets of both abdominal and back muscles (Radebold et al., 2001) and less anticipatory control of the pelvis (Jacobs et al., 2009) illustrate the importance of this region within the impaired robustness in static postural tasks.

Besides postural control in static postures (e.g. standing and sitting), the performance of a dynamic task such as the sit-to-stance-to-sit (STSTS) movement may also be affected in patients with LBP. This movement is demonstrated to be performed on average 60 times a day in a working population (Dall and Kerr, 2010). As a result, optimal movement performance is crucial because of its high daily frequency.

People with LBP demonstrate reduced hip and lumbar range of motion during this task possibly indicating changed pelvic kinematics (Shum et al., 2005). Moreover, energy transfer from the pelvis to the lower limbs is decreased in people with LBP during the sit-to-stand (STS) transfer, resulting in a greater energy demanding task for this people which may exacerbate pain (Shum et al., 2009). These findings suggest changed strategies for the STS movement in people with LBP, but it remains unclear if pain or other underlying mechanisms are responsible for these changes.

Proprioception is a very important neurophysiological capacity in the control of both posture and movement. Altered proprioception is already shown in people with LBP by demonstrating greater repositioning errors in isolated spinal movements (Newcomer et al., 2000; Brumagne et al., 2000; Descarreaux et al., 2005). Moreover, by means of muscle vibration, Brumagne et al. (2008) demonstrated less capacity to upweight proprioceptive feedback from paraspinal muscles to provide optimal standing postural control when the postural task becomes more difficult in people with LBP. Furthermore, Claeys et al. (2011) demonstrated similar conclusions both in standing as well as in sitting, associated with decreased variability in postural control strategy in LBP. These findings suggest that proprioceptive changes or impairments at the lumbar spine observed in people with LBP may also influence the performance of more dynamic total body movements in which the lumbosacral region plays a crucial kinematic role such as the STSTS movement.

Consistent control of the COM is already shown to be important for successful STS performance (Reisman et al., 2002). This optimal control during movements with great mass redistribution (i.e. movements with a moderate to large shift of the COM) could be achieved by optimal pelvic initiation of the movement such as during the sit-up (Cordo and Gurfinkel, 2004). As a result, less lumbar proprioceptive inputs may decrease this optimal pelvic control and thus influence the performance of the STSTS movement. However, although changed kinematics during the STSTS movement are shown, the role of decreased postural control and associated impaired proprioception as a possible underlying mechanism for this altered performance, remains unclear.

Therefore, the general aim of this study was to investigate if proprioceptive impairments demonstrated during static postural tasks in people with LBP are associated with an altered performance of a more dynamic task. As a dynamic task, the STSTS task was chosen because of its clinical usefulness and its good reliability (inter- and intratester) in healthy people as well as in people with LBP (Simmonds et al., 1998). Furthermore, several variables seem to influence the performance of this task (Janssen et al., 2002). Until now, the role of impaired proprioception on the performance of this task remains still obscure. The first specific aim of this study was to investigate if there is an association between altered proprioceptive postural control in people with LBP and the performance of the STSTS movement (dynamic task). The second specific aim was to investigate the role of the pelvis and the trunk in the performance of this dynamic tasks, based on the findings that (1) the lumbosacral region is demonstrated to show impaired proprioceptive capacities during static postural tasks (i.e. standing and sitting) in people with LBP (Brumagne et al., 2008b; Claeys et al., 2011) and (2) the lumbopelvic area plays a crucial kinematic role during the sit-to-stance movement in healthy subjects (Johnson et al., 2009). Muscle vibration, known as a strong stimulus for muscles spindles (Roll and Vedel, 1982), was used to more specifically appraise the proprioceptive steering of the subjects.

2. Methods

2.1.Participants

One hundred and six subjects with LBP and 20 healthy controls voluntarily participated in this study (Table 1). Exclusion criteria were a history of vestibular disorders, neurological or respiratory disease, previous spinal surgery, structural spinal problems, radiculopathy and other musculoskeletal problems (limbs, neck or thorax) at the moment of the test. All subjects had to fill out four questionnaires: a Physical Activity Questionnaire (PAI) (Baecke et al., 1982), the Revised-Oswestry Disability Index (ODI-2) (Fairbank and Pynsent, 2000), the Fear Avoidance Beliefs Questionnaire (Waddell et al., 1993) and the Tampa Scale of Kinesiophobia (TSK) (Vlaeyen et al., 1995). In addition, they had to score the pain at the moment of testing on a numeric rating scale (NRSpain). Subjects were included in the LBP group if they reported a NRSpain> 0 and if they scored ODI> 6 at the moment of testing. However, when these subjects had an acute episode of LBP, they were rescheduled to another testing moment. The healthy controls did not report any pain (NRSpain = 0) and had an ODI score of 0.

All subjects gave their written informed consent and all test procedures were approved by the Biomedical Research Ethics Committee of the KU Leuven with respect to the declaration of Helsinki.

Table 1. Characteristics of the test population.

	Healthy (N=20)	SD	persons with LBP (N=106)	SD	P-value
male	7		25		
female	13		81		
age	18.5	0.5	18.5	0.5	NS
height	170.0	8.5	170.9	9.1	NS
weight	61.0	7.4	63.2	8.5	NS
BMI	21.1	2.4	21.6	2.4	NS
NRS pain (0-10)	**0.0**		**2.0**	**2.2**	**S**
ODI-2 (0-100)	**0.0**		**8.8**	**2.0**	**S**
TSK (17-68)	33.0	6.4	33.1	4.9	NS
FABQPA (0-24)	**4.4**	**5.8**	**7.9**	**5.3**	**S**
FABQW (0-42)	3.0	6.0	4.6	7.6	NS
PAI	8.8	0.8	7.7	1.3	NS

LBP = non-specific low back pain, BMI=Body Mass index, NRS = Numeric Rating Scale, ODI = Oswestry Disability Index, TSK = Tampa Scale for Kinesiophobia, FABQPA = Fear Avoidance Beliefs Questionnaire Physical Activity, FABQW= Fear Avoidance Beliefs Questionnaire Work, PAI = Physical Activity Scale (work index + sport index + leisure-time index, max. score: 5 + 5 + 5=15). NS means not-significant.

2.2.Experimental set-up

Movement analysis

Postural sway characteristics were measured using a six-channel strain gauges force plate (Bertec Corporation, OH, USA). Force plate data were sampled at 500 Hz using a Micro 1401 data-acquisition system and Spike2 software (Cambridge Electronic Design, UK) and low pass filtered with a cutoff frequency of 5 Hz. To evaluate trunk and pelvis position changes in space, two piezo-resistive accelerometers (ICSensors, UK), also connected with the data-acquisition system, were placed on the spinous processes of T1 and S2 vertebra in upright posture.

Muscle vibration and Relative Proprioceptive Weighting (RPW)

The role of proprioception in postural control was directly examined by means of muscle vibration, known as a powerful stimulus of Ia afferents (Roll and Vedel, 1982). The muscle spindles play a crucial role within the proprioceptive system. These proprioceptors are predominantly responsible for signaling position and movement (Proske and Gandevia, 2009). Low amplitude high frequency muscle vibration has been shown to specifically stimulate these proprioceptive afferents (Goodwin et al., 1972; Roll and Vedel, 1982; Brumagne et al., 2000). As a result, larger postural sways during postural control tasks are expected if the CNS uses the signals of the vibrated muscles for optimal postural control (Brumagne et al., 2004; Brumagne et al., 2008a). Thus, subjects mainly rely on ankle muscle proprioceptive signals if they show large posterior postural sways during ankle muscle vibration (i.e. ankle strategy). In contrast, they rely on back muscle proprioceptive inputs if they show anterior postural sways during multifidus muscle vibration (i.e. multi-segmental strategy)

Two self-manufactured muscle vibrators (Maxon motors, Switzerland) were applied bilaterally to triceps surae muscles or to lumbar multifidus muscles, respectively. These muscles were selected, based on previous studies to represent the muscles used in an ankle-steered strategy or a multi-segmental strategy, respectively (Brumagne et al., 2008b). Activation and deactivation of the vibrators was manually controlled. The frequency of the vibration was set at 60 Hz and the amplitude was approximately 0.5 mm. These characteristics of vibration were chosen to induce maximal illusory joint movement and were demonstrated to induce a significant muscle lengthening illusion in healthy individuals (Roll and Vedel, 1982; Cordo et al., 2005). When the CNS is using the signals of the vibrated muscles for postural control, larger directional sways are expected. The direction of the postural sway depends on the reference frame the CNS is using for postural control (Gurfinkel et al., 1995; Brumagne et al., 2008a)

When triceps surae muscles are vibrated during standing, the rigid body above the ankle joint is considered by the CNS as the ‘mobile’ part of the body compared to the ‘stationary’ feet. Triceps surae muscle vibration will induce an illusion of ankle dorsiflexion (lengthening illusion of the m. triceps surae). Consequently, the direction of the sway will be backwards if the CNS uses these proprioceptive signals for postural control to prevent falling. In contrast, lumbar multifidus vibration during standing results in a forward sway if the CNS uses these proprioceptive signals for postural control. The back muscle lengthening illusion corresponds with a posterior pelvic rotation (backward displacement of COM) if the sacrum-pelvis is considered by the CNS as the ‘mobile’ body part compared to the ‘stationary’ legs. Therefore, the CNS will react with an anterior rotation of the pelvis (forward displacement of COM) to prevent falling.’

2.3.Test procedure

All subjects filled out the questionnaires before they performed the tests evaluating proprioception on the force plate. During these tests, they only wore shorts and a bra for the female subjects. To standardize the feet position during the different trials a transparent film (both feet 10 cm separated) was used. All trials had a duration of 60 seconds (Table 2). During the four standing trials (Trials 1, 2, 4 and 5), muscle vibration was initiated 15 seconds after the start of the trial for a duration of 15 seconds. For the STSTS trials (Trials 3 and 6) subjects sat on a stable stool with height adjusted to create a rectangle between the greater trochanter – lateral femoral condyle line and the lateral femoral condyle – lateral malleolus line, respectively. Feet position was standardized using the same transparency sheet from the standing trials. Subjects were asked to adopt a usual sitting posture before performing the movements. Subjects had to perform five STSTS repetitions as fast as possible after a verbal signal (‘start’) by the researcher operating a PC connected to the data-acquisition system.

In all trials vision was occluded and subjects were asked to remain as immobile, but relaxed as possible in upright standing or usual sitting posture, respectively, with the arms hanging loosely along the body.

During Trials 4, 5 and 6, a “foam” condition was used to create a postural condition in which ankle proprioceptive signals were less reliable and therefore the CNS should rely even more on other proprioceptive signals to control posture (Ivanenko et al., 1999).

Table 2. Overview of the different test trials on the force plate

Trial 1	Standing on a stable support with muscle vibration on triceps surae muscles		
	Standing 15 s.	Standing + vibe 15 s.	Standing 30 s.
Trial 2	Standing on a stable support with muscle vibration on multifidus muscles		
	Standing 15 s.	Standing + vibe 15 s.	Standing 30 s.
Trial 3	5 repetitions of the STSTS movement on a stable support as fast as possible		
	Sitting 15 s.	5 repetitions STSTS	Sitting till end of trial
Trial 4	Standing on an unstable support (foam) with muscle vibration on triceps surae muscles		
	Standing 15 s.	Standing + vibe 15 s.	Standing 30 s.
Trial 5	Standing on an unstable support (foam) with muscle vibration on multifidus muscles		
	Standing 15 s.	Standing + vibe 15 s.	Standing 30 s.
Trial 6	5 repetitions of the STSTS movement on an unstable support (foam) as fast as possible		
	Sitting 15 s.	5 repetitions STSTS	Sitting till end of trial

STSTS = sit-to-stance-to-sit

2.4.Data analysis

Displacements of the center of pressure (COP) in anterior-posterior direction were estimated from the raw force plate data using the equation:

$$\mathrm{COP} = \frac{Mx}{Fz}$$

Furthermore, proprioceptive control strategy or Relative Proprioceptive Weighting (RPW) was appraised using the equation:

$$\mathrm{RPW}\frac{TS}{LM} = \frac{(abs\,TS)}{(abs\,TS\ +\ abs\,LM)}$$

In this formula 'abs TS' is the absolute value of the mean COP displacement during triceps surae muscle vibration minus the absolute value of the mean COP displacement during the previbration (baseline) period and 'abs LM' is the absolute value of the mean COP displacement during lumbar multifidus muscle vibration minus the absolute value of the mean COP displacement during the previbration period. A score equal to 1 corresponds to 100% reliance on triceps surae muscle afference. A score equal to 0 corresponds to 100% reliance on lumbar multifidus muscle afference.

In all standing trials (1, 2, 4 and 5), mean values were calculated to appraise the RPW values. During the STSTS trials, the total duration of the five consecutive repetitions as well as the duration of each stance, sit and movement phase was recorded. This subdivision in distinct phases was made based on mean values of the COP: to define the stance phase, the mean value of the COP of during usual standing was used; to define the sit phase, the mean value of the COP of during usual sitting was used (Fig. 1). This use of the force plate data to analyze the duration the STS movement has been demonstrated to be a valid method (Arcelus et al., 2009). To have more insight into the kinematics of the trunk and pelvis, the onsets of the pelvic rotation were recorded by the S2 accelerometer at movement initiation. During the five repetitions, the onsets of pelvic rotation (S2 accelerometer) relative to the onsets of trunk movement (T1 accelerometer) were also recorded.

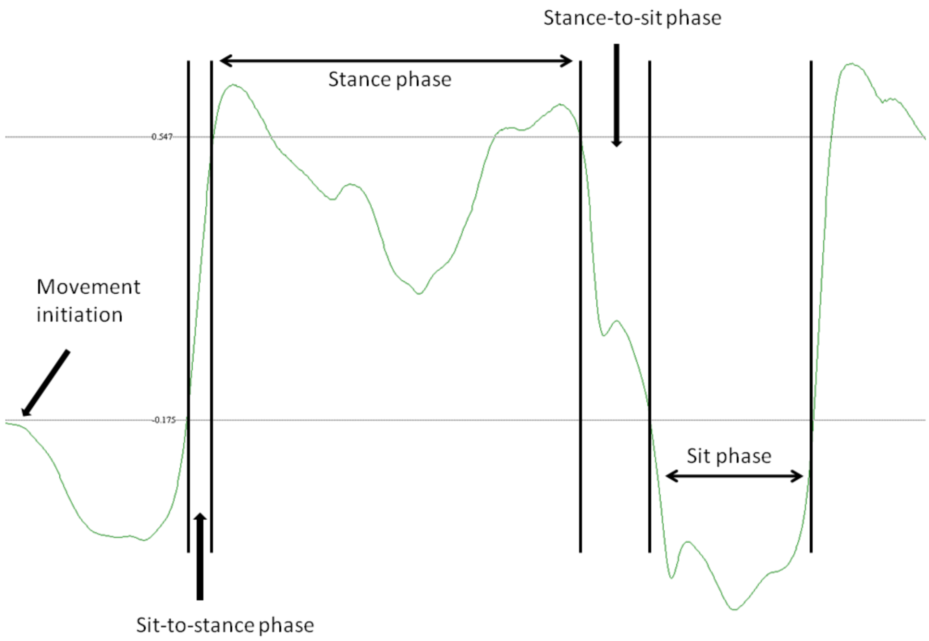


Fig. 1. Raw data of the mean values of the anterior-posterior displacement of the center of pressure (COP) of one sit-to-stance-to-sit (STSTS) movement sequence. Stance and sit phases were defined based on the mean values of the COP during usual standing and usual sitting trials, respectively.

Differences in RPW-values between the conditions, between the trials, and between the LBP and healthy group were compared, based on repeated measures analysis of variance (ANOVA/MANOVA). Furthermore, investigating the STSTS trials, differences in total duration, in duration of the different phases as well as in pelvic rotation onsets between both groups were also calculated based on repeated measures analysis of variance (ANOVA/MANOVA). Where a significant main and interaction effect was found, post hoc tests (Tukey's unequal N HSD) were performed to further analyze the detailed effects. All data are presented as means ± standard deviations (SD). The level of statistical significance was set at $p \leq 0.05$. The statistical analysis was performed with Statistica 9 (Statsoft, OK, USA).

3. Results

3.1.Standing

People with LBP rely more on ankle muscle proprioceptive inputs compared to healthy controls in both standing conditions (RPW stable: healthy 0.63 $\pm$ 0.16 vs. LBP 0.75 $\pm$ 0.16, $p < 0.0001$; RPW unstable: healthy 0.39 $\pm$ 0.13 vs. LBP 0.55 $\pm$ 0.20, $p < 0.0001$). Fig. 2 illustrates the RPW values for both groups in both conditions.

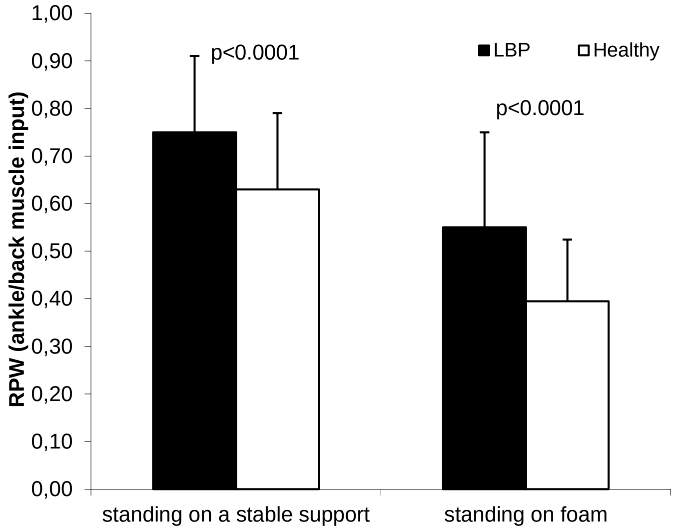


Fig. 2. Results of the Relative Proprioceptive Weighting (RPW) values of the standing trials. Higher RPW-values indicate more reliance on ankle muscle proprioceptive inputs in people with non-specific low back pain (LBP) in both standing conditions.

3.1.1. Sit-to-stance-to-sit

3.1.2. Duration

With feet on a stable support surface, persons with LBP needed significantly more time to perform the five consecutive STSTS movements (LBP: 9.33 s ± 1.49 s vs. healthy: 8.29 s ± 1.23 s; p< 0.005). This longer duration is mainly caused by significantly longer stance phases (LBP: 1.22 s ± 0.20 s vs. healthy: 1.08 s ± 0.15 s; p< 0.05; Fig. 3), while all other phases were not significantly different between the two groups (p> 0.05).

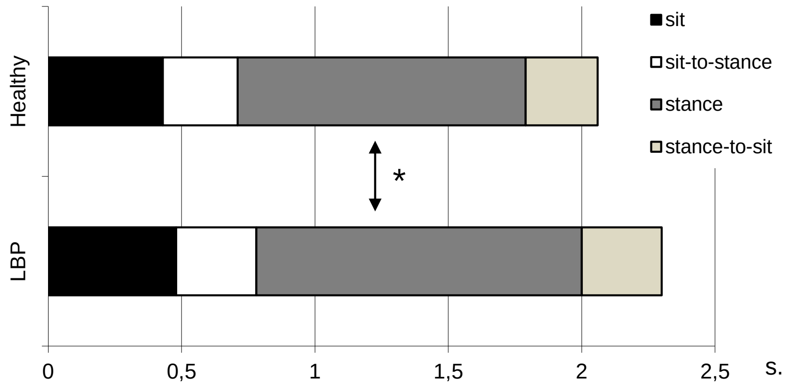


Fig. 3. Mean duration (s) of 1 STSTS movement of both groups with feet on a stable surface, * means a significant difference (P<0.05).

With both feet on an unstable support surface, people with LBP again needed more time compared to the healthy controls to perform the five STSTS repetitions (LBP: 8.96 ± 1.18 s vs. healthy: 8.30 s ± 1.23 s; p< 0.005). This longer performance is caused by both longer stance phases (LBP: 1.15 s ± 0.08 s vs. healthy: 1.03 s ± 0.04 s; p< 0.05) and longer sit phases, while the sit-to-stance and stance-to-sit movements had equal durations in people with LBP and healthy controls (p> 0.05).

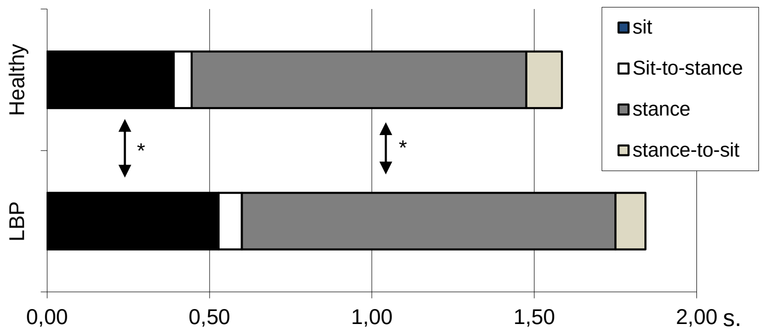


Fig. 4. Mean duration (s) of 1 STSTS movement of both groups with feet on an unstable surface, * means a significant difference (P<0.05).

3.1.3. Pelvic rotation onsets

To have more insight in the pelvic rotation onsets during the performance of the STSTS, onsets of pelvic rotation during the STSTS were recorded by the accelerometers and analyzed. Negative values indicate anticipatory movement of the pelvis; positive values illustrate pelvic movement after the start of the focal movement. There was a significantly later anterior pelvic rotation onset in the LBP group compared to the healthy group in both conditions ($p < 0.005$) to start up the five STSTS repetitions. During the movement sequence, the initiating movement of the pelvis was significantly earlier in the healthy group compared to the LBP group when moving from sit-to-stance on foam (Table 3, figure 5).

Table 3. Anterior pelvic rotation onsets (seconds)

		Healthy N=20		LBP N=106		
Condition	***Phase***	***Mean***	***SD***	***Mean***	***SD***	***P***
Stable	***Start STSTS***	0.10 s.	0.08 s.	0.14 s.	0.14 s.	*S*
Foam	***Start STSTS***	-0.05 s.	0.17 s.	0.14 s.	0.15 s.	*S*
Stable	***Sit-stance***	-0.04 s.	0.01 s.	-0.04 s.	0.00 s.	NS
	Stance-sit	-0.16 s.	0.01 s.	-0.26 s.	0.03 s.	NS
Foam	***Sit-stance***	-0.08 s.	0.09 s.	-0.05 s.	0.06 s.	*S*
	Stance sit	-0.38 s.	0.17 s.	-0.37 s.	0.09 s.	NS

Start STSTS: mean onset of anterior pelvic rotation relative to the initiation of the five repetitions of the sit-to-stance-to-sit movement; Sit-stance: mean onset of anterior pelvic rotation relative to T1 when moving from sit to stance; Stance-sit: mean onset of pelvic movement relative to T1 when moving from stance to sit; P≤0.05 means significant difference (S); p>0.05 means no significant difference (NS).

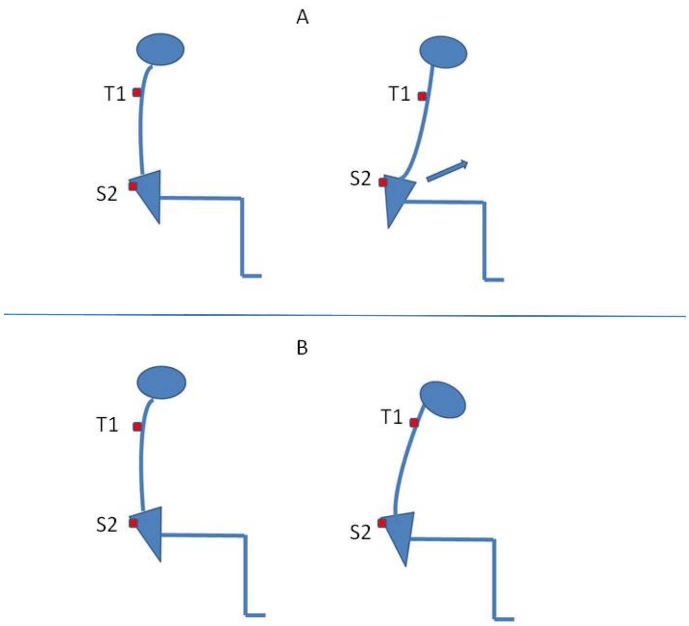


Fig. 5. Graphic representation of A: Earlier onsets of anterior pelvic rotation of a healthy subject to start the STSTS and during the movement sequences (sit-to-stance phase). B: delayed anterior pelvic rotation relative to T1 during the movement sequences.

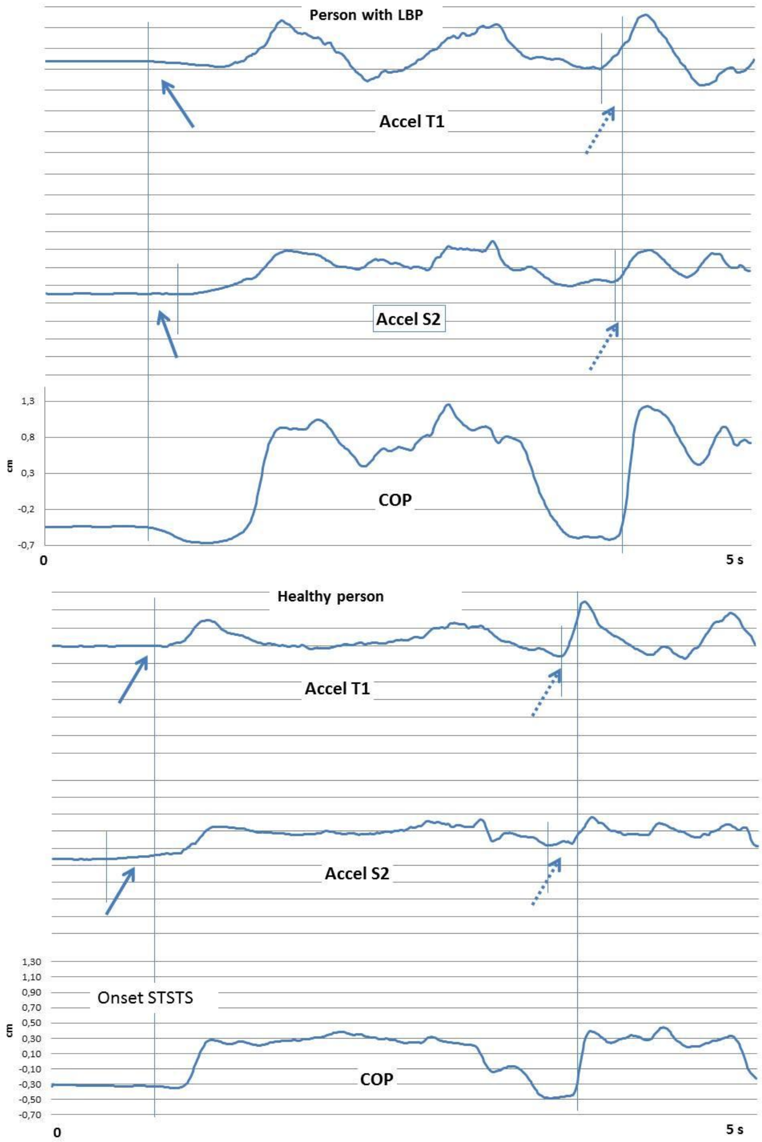


Fig. 6. Raw data file of the pelvic kinematics and the COP trajectories. Healthy people show preparatory pelvic motion during the sit phases. (LBP= low back pain, Accel T1= accelerometer on first thoracic vertebra, Accel S2= accelerometer on second sacral vertebra, COP= center of pressure).

Healthy persons have preparatory pelvic anterior rotation at the onset of the STSTS in contrast to people with LBP, who have no pelvic movement before the onset (solid arrow). When moving from sit to stance during the movement sequence, patients initiate with a lot of thoracic flexion (dotted arrows), in contrast to healthy persons who initiate with anterior pelvic movement.

4. Discussion

The main finding of this study is that people with LBP demonstrated a decreased use of lumbar proprioceptive inputs and performed the STSTS movement significantly slower than healthy subjects presenting with a more optimal proprioceptive control. Different from previous studies analyzing the STSTS (Simmonds et al., 1998; Shum et al., 2005), this study applied a methodological approach to sub-analyze the STSTS in distinct phases (sit, sit-to-stance, stance and stance-to-sit) based on the COP trajectories. Interestingly, this slower performance of the total task was the result of a decrease in speed during the preparatory (transition) phases and not during the focal movement phases. During these preparatory phases (i.e. stance and sit) the direction of the COM of the body switches in the opposite direction.

In our opinion, the additional sub-analysis in distinct phases is important to have more insight into underlying mechanisms of altered postural control and body kinematics during the STSTS in people with LBP. According to Cordo and Gurfinkel (2004) two phases can be distinguished during complex sagittal movements: a preparatory phase and a movement phase. During the preparatory phase, the CNS prepares the body for an optimal movement performance with minimal energy demands during the movement phase (Cordo et al., 2006; Shum et al., 2009). The initiation of pelvic movement to transfer the COM is demonstrated to be crucial during this preparatory phase (Cordo and Gurfinkel, 2004). In the current study, altered pelvic kinematics (i.e. delayed onsets) during these transitional phases were found in the patient group compared to the healthy group. This delay in movement preparation forms the basis for the longer duration of the total movement. Moreover, this also confirms why the STSTS movement is more energy demanding in people with LBP as already demonstrated by Shum et al. (2009). It may be hypothesized that the delay in anterior pelvic rotation initiation results in more trunk flexion during the STSTS movement. Furthermore, repeated flexion combined with mild compressive loads is an important risk factor in the development of intervertebral disc injuries, such as posterior disc herniation (McGill, 2004). The STSTS movement is on average 60 times performed daily (Dall and Kerr, 2010). Consequently, the high frequency of the STSTS movement in combination with the increased flexion-compression component during the movement in subjects with LBP may be a risk factor to develop or maintain spinal pain (Callaghan and McGill, 2001; McGill, 2004). Further (prospective) studies are needed to underscore or refute this hypothesis.

Delayed onsets of deeper abdominal muscle activity is already demonstrated to compromise feedforward lumbopelvic control in people with LBP (Hodges et al., 2003; Jacobs et al., 2009). As a result, the delay in lumbopelvic control may not only compromise postural robustness, but also the performance of total body movements where this region plays a crucial biomechanical role to transfer the COM efficiently (Cordo and Gurfinkel, 2004). A second possible mechanism in the delay of anterior pelvic rotation onsets, may be the decreased proprioceptive afference from the lumbar multifidus as demonstrated during standing and sitting in LBP (Brumagne et al., 2008b; Claeys et al., 2011). The lumbar multifidus plays an important role in the initiation of anterior pelvic rotation (Claus et al., 2009). The delays in pelvic movement initiation were mostly observed in all sit phases (start STSTS stable and foam, sit phase during the movement on foam; Table 3) but not during the stance phases. Moving from sit-to-stance may be a more energy demanding task in which movement preparation by anterior pelvic rotation is more crucial compared to the stance-to-sit movement (Shum et al., 2009).

The presence of pain could be another possible mechanism to explain the delay in onset of anterior pelvic rotation, the longer duration of the sit phase and the associated longer preparatory transition phases. However, in this study, the subjects with LBP had fairly mild pain at the moment of testing (VAS pain=2.0± 2.2). Moreover, patients with an acute episode of pain were rescheduled. Thus, it is unlikely that pain was the main causing factor for these lumbopelvic postural control changes in the current study. Besides altered sensorimotor control, also kinesiophobia, beliefs and decreased physical activity have been proposed as a mechanism associated with changed movement patterns in people with LBP (Hodges and Moseley, 2003). From this perspective it has to be noted that subjects in the current study had very low scores on questionnaires evaluating these factors (Table 1). Furthermore, scores on PAI-index, TAMPA scale for kinesiophobia and FABQW were not statistically different between both groups. Only the score on the FABQPA was statistically significant, but the values on FABQPA were very low. Based on these results, these variables may not clarify the difference in movement pattern between both groups.

The association between a longer duration of the STSTS and altered kinematics of the pelvis demonstrated in this study may be a risk factor to develop or sustain LBP, due to the daily frequently performed STSTS in a working population (Dall and Kerr, 2010). Disrupted motor coordination is demonstrated to be a mechanism in the development of chronic LBP (Panjabi, 2006). However, prospective studies are necessary to clarify the relationship between reduced proprioception in the lumbosacral area and delayed onsets of preparatory pelvic movements in voluntary movements as a mechanism in the development or maintenance of LBP.

The results of this study may have some clinical consequences. First, the rehabilitation of the proprioceptive impairments in the lumbosacral region in people with (mild) LBP needs further attention. Exercises must stimulate people to rely more on back muscle proprioceptive inputs in postural control. Performing postural control exercises on unstable surfaces and in different postural conditions (e.g., standing, sitting, sit-to-stance-to-sit) may be fruitful to stimulate a more multisegmental (including more reliance on lumbar multifidus proprioceptive inputs) proprioceptive postural control strategy. Second, besides exercises in static postural conditions, dynamic movements must be retrained. Lastly, current results suggest to include pelvic control exercises during movements requiring a redistribution of the COM, such as the STSTS. It is hypothesized that optimal pelvic control (e.g. anterior pelvic rotation initiation), especially during preparatory or transition phases of these movements, may improve the performance of this task and could be fruitful in rehabilitation and/or prevention of reoccurrence of LBP.

Despite this novel insight into the STSTS, the current study has some limitations. A first limitation is the use of 2-dimensional instead of 3-dimensional accelerometers. Three-dimensional accelerometers could give additional kinematical information on rotational movements of the pelvis and the trunk. Compensatory rotation in the transverse plane may cause higher loads on spinal tissues and could play an important role in the development of spinal pain (Shum et al., 2007). Second, a more detailed analysis of trunk motion, based on sub-dividing the trunk in different segments (e.g. pelvis, lower lumbar, upper lumbar, lower thoracic, upper thoracic), is essential for understanding the altered trunk motion in people with LBP. Besides changes in pelvic rotation onsets, also other trunk regions may have altered temporal behavior during the STSTS in this population (Johnson et al., 2009). A third limitation may be the characteristics of the test group: a group of young people with mild LBP. Therefore, these results cannot be generalized to older and more disabled patients with LBP. Future studies are necessary in different age groups to have more insight in the role of age as an influencing factor in static and dynamic postural control changes during STSTS. Finally, the cross-sectional study-design does not provide an answer on the cause or result question. Despite the altered performance of a dynamic task in persons with impaired proprioception, current findings do not clarify if proprioceptive changes are a result of LBP or actually cause LBP. Prospective studies investigating proprioception in both static and dynamic postural control conditions are necessary to clarify this research question.

5. Conclusion

Transitioning between sitting and standing is a common daily activity often reported as an aggravating activity in patients with LBP. This study illustrates that in people with mild LBP a suboptimal proprioceptive control strategy is associated with a slower performance of the STSTS. A more detailed analysis showed longer durations of the preparatory (transitional) phases and not during the focal movement phases. During these preparatory phases, delayed onsets of anterior pelvic movement were observed. Results suggest that decreased proprioceptive afference from the lumbar multifidus is associated with these delayed pelvic rotation onsets. In addition, prospective studies are necessary to further clarify if LBP leads to altered proprioceptive postural control and maladaptive dynamic task performance or if altered proprioception and performance of movements may lead to LBP.

6. References

1. Arcelus A, Herry CL, Goubran RA, Knoefel F, Sveistrup H, Bilodeau M. Determination of sit-to-stand transfer duration using bed and floor pressure sequences. IEEE. Trans. Biomed. Eng. 2009;56(10):2485-2492.
2. Baecke JA, Burema J, Frijters JE. A short questionnaire for the measurement of habitual physical activity in epidemiological studies. Am. J. Clin. Nutr. 1982;36(5):936-942.
3. Brumagne S, Cordo P, Lysens R, Verschueren S, Swinnen. The role of paraspinal muscle spindles in lumbosacral position sense in individuals with and without low back pain. Spine 2000;25(8):989-994.
4. Brumagne S, Cordo P, Verschueren S. Proprioceptive weighting changes in persons with low back pain and elderly persons during upright standing. Neurosci. Lett. 2004;366(1):63-66.
5. Brumagne S, Janssens L, Janssens E, Goddyn L. Altered postural control in anticipation of postural instability in persons with recurrent low back pain. Gait. Posture 2008a;28(4):657-662.
6. Brumagne S, Janssens L, Knapen S, Claeys K, Suuden-Johanson E. Persons with recurrent low back pain exhibit a rigid postural control strategy. Eur. Spine J. 2008b;14(9):1177-1184.
7. Callaghan JP, McGill SM. Intervertebral disc herniation: studies on a porcine model exposed to highly repetitive flexion/extension motion with compressive force. Clin. Biomech. 2001;16(1):28-37.
8. Carver S, Kiemel T, Jeka JJ. Modeling the dynamics of sensory reweighting. Biol. Cybern. 2006;95(2):123-134.
9. Claeys K, Brumagne S, Dankaerts W, Kiers H, Janssens L. Decreased variability in postural control strategies in young people with non-specific low back pain is associated with altered proprioceptive reweighting. Eur. J. Appl. Physiol. 2011;111(1):15-123.
10. Claus AP, Hides A, Moseley GL, Hodges PW. Different ways to balance the spine: subtle changes in sagittal spinal curves affect regional muscle activity. Spine 2009;34(6):E208-E214.

11. Cordo PJ, Gurfinkel VS. Motor coordination can be fully understood only by studying complex movements. Prog. Brain Res. 2004;143:29-38.
12. Cordo PJ, Gurfinkel VS, Brumagne S, Flores-Vieira C. Effect of slow, small movement on the vibration-evoked kinesthetic illusion. Exp. Brain Res. 2005;167(3):324-334.
13. Cordo PJ, Hodges PW, Smith TCS, Brumagne S, Gurfinkel VS. Scaling and non-scaling of muscle activity, kinematics, and dynamics in sit-ups with different degrees of difficulty. J. Electromyogr. Kinesiol. 2006;16(5):506-521.
14. Dall PM, Kerr A. Frequency of the sit to stand task: An observational study of free-living adults. Appl. Ergon. 2010;41(1):58-61.
15. Descarreaux M, Blouin JS, Teasdale N. Repositioning accuracy and movement parameters in low back pain subjects and healthy control subjects. Eur. Spine J. 2005;14(2):185-191.
16. Fairbank JC, Pynsent PB. The Oswestry Disability Index. Spine 2000;25(22):2940-2952.
17. Goodwin GM, McCloskey DI, Matthews PB. Proprioceptive illusions induced by muscle vibration: contribution by muscle spindles to perception. Science 1972;175(28):1382-1384.
18. Gurfinkel VS, Ivanenko Y, Levik Y, Babakova IA. Kinesthetic reference for human orthograde posture. Neuroscience 1995;68(1):229-243.
19. Henry SM, Hitt JR, Jones SL, Bunn JY. Decreased limits of stability in response to postural perturbations in subjects with low back pain: Clin. Biomech. 2006;21(9):881-892.
20. Hodges PW, Moseley GL. Pain and motor control of the lumbopelvic region: effect and possible mechanisms. J. Electromyogr. Kinesiol. 2003;13(4):361-370.
21. Hodges PW, Moseley GL, Gabrielsson A, Gandevia SC. Experimental muscle pain changes feedforward postural responses of the trunk muscles. Exp. Brain Res. 2003;151(2):262-271.
22. Ivanenko YP, Talis VL, Kazennikov OV. Support stability influences postural responses to muscle vibration in humans. Eur. J. Neurosci. 1999;11(2):647-654.

23. Jacobs JV, Henry SM, Nagle KJ. People with chronic low back pain exhibit decreased variability in the timing of their anticipatory postural adjustments. Behav. Neurosci. 2009;123(2):455-458.
24. Janssen WG, Bussmann HB, Stam HJ. Determinants of the sit-to-stand movement: a review. Phys. Ther. 2002;82(9):866-879.
25. Johnson MB, Cacciatore TW, Hamill J, Emmerik RE. Multi-segmental torso coordination during the transition from sitting to standing. Clin. Biomech. 2009;25(3):199-205.
26. McGill SM. Linking latest knowledge of injury mechanisms and spine function to the prevention of low back disorders. J. Electromyogr. Kinesiol. 2004;14(1):43-47.
27. Mientjes MI, Frank JS. Balance in chronic low back pain patients compared to healthy people under various conditions in upright standing. Clin. Biomech. 1999;14(10):710-716.
28. Mok NW, Brauer SG, Hodges PW. Hip strategy for balance control in quiet standing is reduced in people with low back pain. Spine 2004;29(6):E107-E112.
29. Mok NW, Brauer SG, Hodges P W. Failure to use movement in postural strategies leads to increased spinal displacement in low back pain. Spine 2007;32(19):E537-E543.
30. Newcomer K L, Laskowski ER, Yu B, Johnson JC, An KN. Differences in repositioning error among patients with low back pain compared with control subjects. Spine 2000;25(19):2488-2493.
31. Panjabi MM. A hypothesis of chronic back pain: ligament subfailure injuries lead to muscle control dysfunction. Eur. Spine J. 2006;15(5):668-676.
32. Proske U, Gandevia SC. The kinaesthetic senses. J. Physiol. 2009;587(17):4139-4146.
33. Radebold A, Cholewicki J, Polzhofer GK, Greene HS. Impaired postural control of the lumbar spine is associated with delayed muscle response times in patients with chronic idiopathic low back pain. Spine 2001;26(7):724-730.
34. Reisman DS, Scholz JP, Schoner G. Coordination underlying the control of whole body momentum during sit-to-stand. Gait. Posture 2002;15(1):45-55.

35. Roll JP, Vedel JP. Kinaesthetic role of muscle afferents in man, studied by tendon vibration and microneurography. Exp. Brain Res. 1982;47(2):177-190.
36. Shum GL, Crosbie J, Lee RY. Effect of low back pain on the kinematics and joint coordination of the lumbar spine and hip during sit-to-stand and stand-to-sit. Spine 2005;30(17):1998-2004.
37. Shum GL, Crosbie J, Lee RY. Three-dimensional kinetics of the lumbar spine and hips in low back pain patients during sit-to-stand and stand-to-sit. Spine 2007;32(7):E211-E219.
38. Shum GL, Crosbie J, Lee RY. Energy transfer across the lumbosacral and lower-extremity joints in patients with low back pain during sit-to-stand. Arch. Phys. Med. Rehabil. 2009;90(1):127-135.
39. Simmonds MJ, Olson SL, Jones S, Hussein T, Lee CE, Novy D, Radwan H. Psychometric characteristics and clinical usefulness of physical performance tests in patients with low back pain. Spine 1998;23(22):2412-2421.
40. Vlaeyen JW, Kole-Snijders AM, Boeren RG, van Eek H. Fear of movement/(re)injury in chronic low back pain and its relation to behavioral performance. Pain 1995;62(3):363-372.
41. Waddel G, Newton M, Henderson I, Somerville D, Main C J. A Fear-Avoidance Beliefs Questionnaire (FABQ) and the role of fear-avoidance beliefs in chronic low back pain and disability. Pain 1993;52(2):157-168.

Chapter 4

Sagittal evaluation of usual standing and sitting spinal posture

Submitted to *Journal of Bodywork and Movement Therapies*

Kurt Claeys[1,2] PT, MT; Simon Brumagne[1] PhD, PT; Jacques Vanderhaeghen[2] PhD, MSc; Jan Deklerck[2] MSc.; Wim Dankaerts[1] PhD, PT, MT

1. Research group Musculoskeletal Rehabilitation, KU Leuven Department of Rehabilitation Sciences, University of Leuven, Leuven, Belgium
2. Department Health Care, Catholic University College of Bruges-Ostend, Xaverianenstraat 10, B-8200 Brugge, Belgium

Abstract

Postural rehabilitation often plays an important role in the management of non-specific low back pain. While cervical and lumbar inter-correlations have been demonstrated previously, the different role of the pelvis and the thoracic spine for postural control in sitting and standing remains unclear. The aim of the current study was to investigate postural inter-correlations between all spinal regions in usual standing and sitting. Based on digital photographs eight postural angles (lumbar curve, thoracic inclination, trunk angle, pelvic tilt, lumbar angle, head angle, neck angle and cervicothoracic angle) were analyzed in 99 young healthy persons. Pearson inter-correlations between adjacent and non-adjacent postural angles were calculated. In usual sitting pelvic tilt demonstrated mostly medium inter-correlations with five out of seven other postural angles, compared to three in usual standing. In usual standing trunk angle showed five out of seven mostly medium inter-correlations with other regions compared to four out of seven in usual sitting. Usual sitting and usual standing posture are two different positions: a different base support and a different hip angle result in different postural sagittal inter-correlations. The weak correlations suggest a large between-subject variability in sagittal spinal posture, without the existence of one optimal sagittal posture.

Key words: posture, spine, postural inter-correlations

1. Introduction

Since alterations of spinal curvatures during static standing and sitting postures have been demonstrated to be associated with higher mechanical loading, neutral spinal alignment is regarded as optimal loading. Indeed, it has been suggested that non-neutral spinal postures may play a role in the development and maintenance of postural related spinal pain. Silva et al. (2009) demonstrated that an increase in forward head posture during standing was associated with chronic non-traumatic neck pain. Chronic low back pain was associated with increased lumbar lordosis, more anterior pelvic rotation (Evcik and Yucel, 2003) and patellofemoral joint pain (Tsuji et al., 2002). Different usual sitting postures were identified between subgroups of patients with chronic low back pain (patients with an active extension pattern or a flexion pattern) and healthy controls (Dankaerts et al., 2006). In an older population, increase in thoracic kyphosis was associated with increased incidence of intrascapular pain, next to increased body sway, gait unsteadiness and higher risk of falls (Fon et al., 1980; Griegel-Morris et al., 1992; Sinaki et al., 2005).

Previously, four standing postures have been described based on sagittal X-rays: a ‘neutral’ (or ‘optimal’) posture, a ‘hyperlordotic’ posture (lumbar lordosis and thoracic kyphosis), a ‘flat back’ (flattened lumbar and thoracic curves) and a ‘sway back’ posture (backward displacement of the thoracic relative to the pelvis) (Kendall F.P. et al., 1993). Subdivision into these postural types was based both on pelvic orientation and the rate of kypholordosis of the thoracolumbar spine. However, correlations between the different spinal regions for the several subtypes remained unclear. More recently, this subdivision was also demonstrated using sagittal digital photographs in combination with external markers on anatomical bony points (Smith et al., 2008). In this study, neutral spinal postures were less associated with LBP. The pelvic orientation plays an important role for sagittal spinal curvature in standing: pelvic anteversion may be associated with more lumbar lordosis, pelvic retroversion may be related to less lumbar lordosis and possibly a more forward position of C7 relative to the sacrum (Roussouly and Pinheiro-Franco, 2011).

For both standing (Dunk et al., 2005; Kuo et al., 2009; O'Sullivan et al., 2006b) and sitting (Black et al., 1996; Kuo et al., 2009; Sprigle et al., 2002), a method of using external markers in combination with digital photographs to analyze sagittal posture was shown to be reliable and valid in postural evaluation. Using this research methodology, significant inter-correlations between the lumbopelvic and the cervical region in end-range sitting postures (i.e. erect and slouched sitting) were demonstrated: a more lumbar extended position was correlated with a more flexed cervical spine (mid and lower); in contrast, more lumbar flexion correlated with more cervical extension (Black et al., 1996). However, variation in movement between upper and lower cervical spine among subjects was demonstrated and the inter-correlations between the pelvis and the neck were significant, but very small (Black et al., 1996). Furthermore, the role of the spinal regions between (i.e. lumbar and thoracic) was not evaluated in this study and remained unclear. More recently, it was demonstrated that more thoracic kyphosis correlated with more upper cervical extension in sitting as well as in standing (O'sullivan et al., 2002; Straker et al., 2008). Kuo et al. (2009) were the first to investigate postural inter-correlations of all spinal regions in both usual standing and sitting. However, only correlations between adjacent spinal regions were reported. As a consequence, the magnitude of the spinal interaction between the lumbopelvic region (pelvis, lumbar spine) and the cervical spine remains unknown.

While postural rehabilitation often plays an important role in contemporary clinical management of spinal problems, there is still a paucity of studies investigating spinal inter-correlations during commonly adopted postures such as usual standing and sitting. As a result, the aim of the current study was to investigate the postural inter-correlations between two spinal angles (pelvic tilt and the trunk angle) and all other spinal regions (lumbar, thoracic, cervical and head) in usual standing and usual sitting.

2. Methods

2.1. Subjects

A total of 99 subjects (25 men and 74 women) without serious spinal problems were recruited to voluntarily participate in this study. Participants were first year physiotherapy students and confirmed to have no recent spinal (cervical, thoracic or lumbar) pain. All subjects gave their written informed consent. Test procedures were approved by the Medical Research Ethics Committee of KU Leuven with respect to the declaration of Helsinki (Ethical Principles for Medical Research Involving Human Subjects). Table 1 shows the characteristics of the subjects.

Table 1. Characteristics of the subjects

	Healthy	
	Male	Female
Number	25	74
Age (+/-SD)	19.6 yr (+/-1.6)	
Weight (+/-SD)	64.3 kg (+/-8.9)	
Height (+/-SD)	171.4 cm (+/-7.9)	
BMI (+/-SD)	21.89 kg/m^2 (+/-2.3)	

SD = standard deviation, BMI = body mass index,

2.2. Instrumentation and methods

Prior to data collection, photo-reflective markers were attached on the right side of nine bony points of each subject: just lateral of the eye, just anterior of the ear, lateral tip of acromion, spinous process of the cervical vertebra C7, spinous process of the thoracic vertebra T12, spinous process of the lumbar vertebra L3, spinous process of the sacrum S2, anterior superior iliac spine (ASIS), greater trochanter (midpoint).

After the placement of the markers digital photographs were taken from the usual standing and usual sitting posture of the subject. For the usual standing position, subjects were asked to stand as usual, with their feet 10 cm apart, with their gaze horizontally and with both arms loosely along their body. To optimize visibility of the markers on the pelvis and trochanter the right elbow was passively flexed by the investigator without moving other regions. For usual sitting, a height adjustable stool was used. Subjects were positioned with a 90 degree angle between femur and tibia. The position of the arms and feet was the similar as in the standing position.

A digital photo camera (Sony A200 DSLR-A200K scope DT18-70mm F3.5-5.6) attached to a tripod with a height of 0,94 meter and 6 meter away from the subject, was used to make the photographs. Figure 1 shows an overview of all postural angles; Figure 2 and 3 illustrate both postural positions.

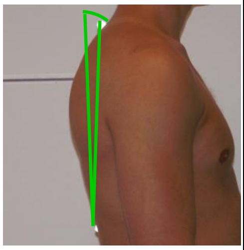
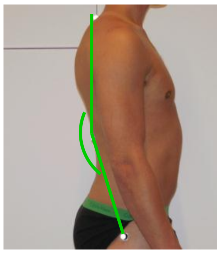
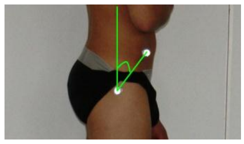
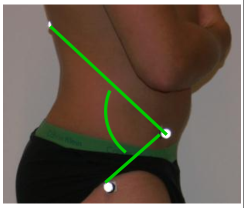
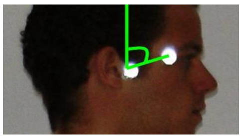
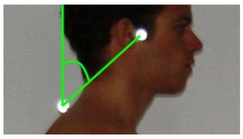
Lumbar curve (T12–L3–S2) 	Thoracic inclination (C7–T12–Vertical) 	Trunk angle (C7–T1–Trochanter) 	Pelvic tilt (ASIS–Trochanter–Vertical) 
Lumbar angle (T12–ASIS–Trochanter) 	Head angle (Eye–Ear–Vertical) 	Neck angle (Ear–C7–Vertical) 	Cervicothoracic angle (Ear–C7–T12 

Fig. 1. The postural angles

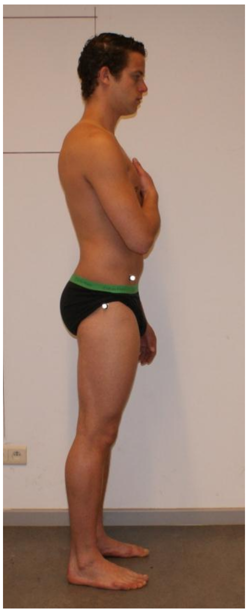


Fig. 2. Usual standing posture.

Fig. 3. Usual sitting posture

To calculate the different postural angles all photographs were imported into MATLAB r2008a (MathWorks Inc., Massachusetts USA) and the co-ordinates of each marker were determined manually. Subsequently, all co-ordinates were imported in MathCAD 14 (PTC, USA). Next, the nine postural angles were calculated by MathCAD 14 (PTC, USA) using formulas of trigonometry. These angles have been described in previous studies investigating spinal posture (Smith et al., 2008; Straker et al., 2008). The use of these markers in combination with digital photographs has been demonstrated to be a reliable method for postural research (Black et al., 1996).

2.3. Statistical analysis

To evaluate the inter-correlations between cervical, thoracic, lumbar and pelvic angles in both postural conditions, Pearson correlation coefficients were calculated using statistical software SPSS 17 (SPSS Inc. Chicago, IL). The level of significance was set at $p < 0.05$. Based on Cohen (1988), following values were used to interpret the correlations: no correlation = (-)0.09 to 0.0; small correlation = (-)0.1 to (-)0.3; medium correlation = (-)0.3 to (-)0.5; strong correlation = (-)0.5 to (-)1.

3. Results

Table 2 gives an overview of all mean values of the different postural angles in both postural conditions for both groups.

Table 2. Mean postural angles (+/-SD) in degrees for the 2 postural conditions

Angle	Usual standing	Usual sitting
Lumbar curve	155,3 ± 10,9	168,4 ± 22,4
Thoracic inclination	6,3 ± 10,8	16,1 ± 16,9
Trunk angle	205,8 ± 10,3	226,4 ± 12,3
Pelvic tilt	38,3 ± 8,9	19,8 ± 15,3
Lumbar angle	93,4 ± 13, 3	119,9 ± 21,7
Head angle	69,6 ± 12,0	70,2 ± 16,5
Neck angle	44,0 ± 8,3	48,7 ± 11,7
Cervicothoracic angle	137,2 ± 11,6	134,9 ± 27,9

Pelvic tilt has small to medium inter-correlations with all other spinal angles during sitting: an increase in anterior pelvic tilt correlates with increased lumbar lordosis (r= -0.3, p< 0.05), increased thoracic inclination (r= 0.4, p< 0.005) and more forward head position (decreased cervicothoracic angle; r= -0.4, p< 0.005, increased thoracic flexion; r= 0.4, p< 0.005). In usual standing, no significant correlation between the pelvic tilt and the lumbar curve, thoracic flexion and the cervicothoracic angle were present. These results indicate a clear postural influence of the pelvis on all other spinal regions in usual sitting, which is to a lesser extent present in usual standing. Furthermore, the directional influence of increased pelvic tilt changes when moving from usual sitting to usual standing: increased anterior pelvic tilt correlates with decreased neck angle (r=-0.2, p<0.05) and decreased head angle (r=-0.3, p<0.05) in usual standing, which is reversed in usual sitting. Figure 4 gives an overview of the inter-correlations between the pelvic tilt/trunk angle and all other spinal angles for both postural conditions.

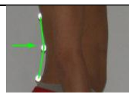
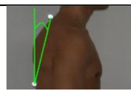
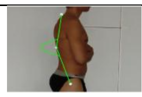
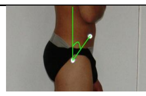
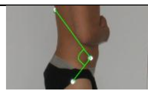
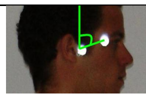
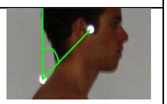
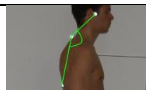
Correlation with pelvic tilt 		Correlation with trunk angle 		Postural angle
Usual sitting	Usual standing	Usual sitting	Usual standing	
-0.3* (M)	0.0 (N)	-0.0 (N)	0.1 (S)	
0.4** (M)	-0.1 (S)	-0.4** (M)	0.5** (STR)	
-0.2* (S)	-0.4** (M)	-	-	
-	-	-0.2* (S)	-0.4** (M)	
-0.3** (M)	-0.8** (STR)	0.3** (M)	0.4** (M)	
0.3** (M)	-0.3* (M)	-0.2* (S)	0.1 (S)	
0.2* (S)	-0.2* (S)	-0.4** (M)	0.5** (STR)	
-0.4** (M)	-0.1 (S)	0.6** (STR)	0.3** (M)	

Fig. 4. Correlations between pelvic tilt/trunk angle and all other spinal angles for both postural conditions

* p<0.05; ** p<0.005; N = no correlation; S = small correlation; M = medium correlation; STR = strong correlation

Different directional behavior between usual standing and usual sitting was also demonstrated between the thoracolumbar spine and the cervical region: an increased trunk angle correlates with decreased neck angle ($r= -0.4$, $p< 0.005$) and decreased head angle ($r= 0.2$, $p< 0.05$) in sitting. In contrast, in usual standing increased trunk angle correlates with increased neck angle ($r= 0.5$, $p< 0.005$). Moreover, increased trunk angle correlates with decreased thoracic inclination in usual sitting ($r= -0.4$, $p< 0.005$) and increased thoracic inclination in usual standing ($r= 0.5$, $p< 0.005$).

4. Discussion

The main findings of this study are the presence of postural inter-correlations between adjacent and non-adjacent spinal regions. These inter-correlations differ in magnitude and direction between usual standing and sitting. In usual sitting, the pelvic tilt has most (5/7) medium inter-correlations with other spinal regions, except for the trunk and neck angle. In contrast, in usual standing the trunk angle has most (5/7) medium (3/7) to strong (2/7) correlations with other spinal regions, except the lumbar curve and the head angle. It must be noticed that only 4 correlations out of 28 were calculated as strong correlations according to Cohen (1988). Eleven out of 28 correlations were calculated as 'no' of 'small' correlation (Figure 4).

4.1.Correlation with pelvic tilt

An increase in pelvic tilt correlates with more lumbar lordosis in usual sitting, but not in usual standing (Figure 4). This finding is in contrast with other studies who demonstrated moderate to high correlations between pelvic and lumbar angles in usual standing (Kuo et al., 2010; Roussouly and Pinheiro-Franco, 2011; Vialle et al., 2005). Methodological differences in evaluating pelvic orientation may explain the differences with the current study and previous studies: firstly, Kuo et al. measured pelvic inclination by placing markers on S2 and the ASIS, while Vialle et al (2005) used X-rays to calculate the sacral orientation. Although the leg position was standardized in the current study, different (transversal) hip angles may influence the results of this pelvic position. Pelvic tilt was evaluated by placing makers on the ASIS and the greater trochanter. As a result, standing with more hip internal rotation may result in less pelvic anterior rotation compared to standing with hip external rotation despite unchanged pelvic orientation. Secondly, in the current study a young population was tested (19.1 ± 1.1 yr). In contrast, the age of the subjects in the study by Kuo et al. (2009) ranged from 17 – 83 years. Kuo et al. (2009) demonstrated significant differences in some postural angles between different age groups. As a result, it is reasonable to hypothesize that postural correlations are influenced by age. Secondly, differences between studies and the mostly small to medium correlations suggest within-subject variability in sagittal spinal alignment. However, the demonstrated findings of a more anteriorly tilted pelvis correlating with a more upright thoracic, cervical and cervicothoracic spine may have some consequences for postural re-education. Despite the mostly small to correlations, optimizing pelvic posture may help to facilitate lumbar, thoracic and cervical posture.

4.2.Correlation with the trunk angle

In usual standing, an increased trunk angle correlates with an increase in both, neck angle and cervicothoracic angle (Figure 4). These regional inter-correlations suggest that people who have an increased kypholordosis also tend to flex the mid and lower cervical spine (forward head posture) and increase the flexion in thoracic spine (increased trunk angle). While this was not under investigation in this study, it is hypothesized that this may be a strategy to have fixed gaze.

In usual sitting, an increased trunk angle correlates with a decrease in head and neck angles. These inter-correlations suggest a compensatory postural correction at the lower, mid and upper cervical spine to keep the gaze forward. When evaluating the lumbosacral area in sitting, there is no association between the trunk angle and the lumbar curve and a negative correlation between the trunk angle and the pelvic tilt (Figure 4). These findings illustrate a more postural steering role of the pelvis in spinal orientation in sitting compared to standing.

In standing, it is also possible to make small corrections at the lower limbs to keep the center of gravity within the base support. This mechanism disappears in a sitting position, where correction of the center of gravity to maintain equilibrium is likely controlled by the pelvis.

4.3. General remarks regarding the correlations

Despite some clear correlations between regions, caution is required to take clear conclusions about integrating these results in postural rehabilitation. Correlations are often small to medium, which illustrates a clear between-subject variability. Different spinal alignments may suffice and one optimal spinal alignment with a clear steering region may not exist. However, in rehabilitation, we cannot ignore the role of optimizing posture in some patients. Despite the weak correlations, this study shows that postural corrections may be initiated with optimizing pelvic posture in sitting. In contrast, in standing positions, pelvic corrections do not suffice. In this standing position, optimizing the trunk position may facilitate the cervical posture.

4.4.Limitations and directions for future investigations

Results of current study cannot be generalized to the whole young population without spinal pain, although some postural correlations between spinal regions are demonstrated. Therefore, the test group is too small and the range of age of the subjects is too limited. There is more need for clinical studies evaluating postural correlations in both in healthy people as well as in specific subgroups of patients with spinal problems. The focus should be on these patients with spinal pain with a clear aggravating and easing postural link.

The current study demonstrating a moderate steering role for the trunk angle in standing and the findings of Roussoly and Nnadi (2010) illustrate that the thoracic spine should be investigated more in detail in postural research. Previous research demonstrated the need to differentiate the lumbar region in an upper and lower lumbar spine, with different kinematic properties and different inter-correlations between postures (Mitchell et al., 2008). Based on these findings it could also be hypothesized that in the thoracic region a subdivision in upper and lower thoracic curve is likely required to further clarify the postural inter-correlations between spinal regions. Especially since in the standing position, correlations with the trunk angle and other spinal regions are more distinct than the correlations with the pelvic tilt. It could be hypothesized that in the sitting position, with broad support base, pelvic adjustments may be more adequate to keep the center of mass of the trunk and the neck within the support base. However, during standing, pelvic postural corrections may be not sufficient enough to keep the center of mass of the legs, trunk and neck within the narrow support base. As a result, postural adjustments at the pelvis only are not sufficient. Consequently, postural corrections also occur at the thoracolumbar spine.

Another limitation of this study is the fact that no subgrouping based on gender and BMI was made. Gender may influence postural inter-correlations because different postures in male and female population have already been observed. Similar, BMI may affect postural inter-correlations since it is demonstrated that obesity may influence standing posture (Black et al., 1996; Dunk et al., 2005; Edmondston et al., 2011; Mitchell et al., 2008). In the current study, the average BMI was under 25, so the results of the current study can only be generalized for a specific population with a BMI < 25.

To have more insights in the kinematics of the spine and the inter-correlations between spinal regions during functional activities, dynamic tasks (such as lifting and bending) should be evaluated. Likely, there may exist some specific postural inter-correlations as demonstrated in this study during these functional movements. More insights in static and dynamic postures and their postural inter-correlations may be very useful for clinical practice.

Investigating postural correlations in combination with EMG may clarify the loading on the spine during the postural tasks. Some patients have quite neutral spinal curvatures in sitting but develop more loading due to higher muscular activity of the superficial back muscles (O'Sullivan et al., 2006a). Higher (muscular) loadings may be a mechanism in the development of spinal pain.

Despite several studies already investigated spinal posture using external markers, currently there is still substantial variety in placement of the markers and the different postural angles evaluated. So to optimize postural research and to facilitate comparison of the results between different studies there is a need to a more standardized method for spinal postural analysis.

5. Conclusion

Differences in postural inter-correlations between adjacent and non-adjacent spinal postural angles in standing and sitting were demonstrated. The mostly small to medium postural correlations in the current study reflect a between-subject variability. A lot of variables may influence how people align their spinal regions: base support, mass distribution en other anthropometric characteristics Future clinical studies should evaluate static and dynamic postural correlations in specific subgroups of patients with postural spinal pain.

6. References

1. Black KM, McClure P, Polansky M. The influence of different sitting positions on cervical and lumbar posture. Spine1996;21(1):65-70.
2. Cohen, J,. Statistical power analysis for the behavioral sciences (2nd ed.), 1988;Lawrence Erlbaum Associates.
3. Dankaerts W, O'Sullivan P, Burnett A, Straker L. Differences in sitting postures are associated with nonspecific chronic low back pain disorders when patients are subclassified. Spine 2006;31(6):698-704.
4. Dunk NM, Lalonde J, Callaghan JP. Implications for the use of postural analysis as a clinical diagnostic tool: reliability of quantifying upright standing spinal postures from photographic images. J. Manipulative Physiol. Ther. 2005;28(6):386-392.
5. Edmondston SJ, Waller R, Vallin P, Holthe A, Noebauer A, King E. Thoracic spine extension mobility in young adults: influence of subject position and spinal curvature. J. Orthop. Sports Phys. Ther. 2011;41(4):266-273.
6. Evcik D, and Yucel A. Lumbar lordosis in acute and chronic low back pain patients. Rheumatol. Int. 2003;23(4):163-165.
7. Fon GT, Pitt MJ, Thies AC. Thoracic kyphosis: range in normal subjects. Am. J. Roentgenol. 1980;134(5):979-983.
8. Griegel-Morris P, Larson K, Mueller-Klaus K, Oatis CA. Incidence of common postural abnormalities in the cervical, shoulder, and thoracic regions and their association with pain in two age groups of healthy subjects. Phys. Ther. 1992;72(6):425-431.
9. Harbourne RT, and Stergiou N. Nonlinear analysis of the development of sitting postural control. Dev. Psychobiol. 2003;42(4):368-377.
10. Harbourne RT, and Stergiou N. Movement variability and the use of nonlinear tools: principles to guide physical therapist practice. Phys. Ther. 2009;89(3):267-282.
11. Kendall FP, McCreary EK, Provance PG. Muscles: Testing and Function, With Posture and Pain (4th ed.). 1993;Williams & Wilkins, Baltimore.

12. Kuo YL, Tully EA, Galea MP. Video analysis of sagittal spinal posture in healthy young and older adults. J. Manipulative Physiol. Ther. 2009;32(3):210-215.
13. Kuo YL, Tully EA, Galea MP. Kinematics of sagittal spine and lower limb movement in healthy older adults during sit-to-stand from two seat heights. Spine 2010;35(1): E1-E7.
14. Mitchell T, O'sullivan PB, Burnett AF, Straker L, Smith A. Regional differences in lumbar spinal posture and the influence of low back pain. BMC. Musculoskelet. Disord.2008;9:152.
15. O'Sullivan P, Dankaerts W, Burnett A, Chen D, Booth R, Carlsen C, Schultz A. Evaluation of the flexion relaxation phenomenon of the trunk muscles in sitting. Spine 2006a;31(17):2009-2016.
16. O'Sullivan P, Dankaerts W, Burnett A, Straker L, Bargon G, Moloney N, Perry M, Tsang S. Lumbopelvic kinematics and trunk muscle activity during sitting on stable and unstable surfaces. J. Orthop. Sports Phys. Ther. 2006b;36(1):19-25.
17. O'sullivan PB, Grahamslaw KM, Kendell M, Lapenskie SC, Moller NE, Richards KV. The effect of different standing and sitting postures on trunk muscle activity in a pain-free population. Spine 2002;27(11):1238-1244.
18. Roussouly P, and Pinheiro-Franco JL. Biomechanical analysis of the spino-pelvic organization and adaptation in pathology. Eur. Spine J.2011.DOI 10.1007/s00586-011-1928-x.
19. Sinaki M, Brey RH, Hughes CA, Larson DR, Kaufman KR. Balance disorder and increased risk of falls in osteoporosis and kyphosis: significance of kyphotic posture and muscle strength. Osteoporos. Int. 2005;16(8):1004-1010.
20. Smith A, O'Sullivan P, Straker L. Classification of sagittal thoraco-lumbo-pelvic alignment of the adolescent spine in standing and its relationship to low back pain. Spine 2008;33(19):2101-2107.
21. Sprigle S, Wootten M, Bresler M, Flinn N. Development of a noninvasive measure of pelvic and hip angles in seated posture. Arch. Phys. Med. Rehabil. 2002;83(11):1597-1602.

22. Straker LM, O'sullivan PB, Smith AJ, Perry MC. Relationships between prolonged neck/shoulder pain and sitting spinal posture in male and female adolescents. Man. Ther. 2009;14(3);321-329.
23. Tsuji T, Matsuyama Y, Goto M, Yimin Y, Sato K, Hasegawa Y, Ishiguro N. Knee-spine syndrome: correlation between sacral inclination and patellofemoral joint pain. J. Orthop. Sci. 2002;7(5):519-523.
24. Vialle R, Levassor N, Rillardon L, Templier A, Skalli W, Guigui P. Radiographic analysis of the sagittal alignment and balance of the spine in asymptomatic subjects. J. Bone Joint Surg. Am. 2005;87(2):260-267.

Chapter 5

Young individuals with a more ankle-steered proprioceptive control strategy may develop mild non-specific low back pain

Submitted to *Journal of Electromyography and Kinesiology*

Kurt Claeys[1,2], Wim Dankaerts[1], Lotte Janssens[1], Madelon Pijnenburg[1], Simon Brumagne[1]

1. Research group Musculoskeletal Rehabilitation, KU Leuven Department of Rehabilitation Sciences, University of Leuven, Leuven, Belgium
2. Department Health Care, Catholic University College of Bruges-Ostend, Bruges, Belgium

Abstract

Altered proprioceptive postural control has been demonstrated in people with non-specific low back pain (LBP). However, the cause-effect relation remains unclear. Therefore, more prospective studies are necessary.

Proprioceptive postural control of 104 subjects was evaluated at baseline using a force plate and with application of vibration stimulation on ankle and back muscles. Spinal postural angles were measured with digital photographs. Psychosocial variables and physical activity was registered using questionnaires. Ninety subjects were followed over two years concerning their LBP status, 14 persons decided not to fulfill the two year follow-up.

Four distinct groups have been determined after two years based on pain and disability scores: never LBP, no LBP at intake with future mild LBP, mild LBP at intake with no further LBP, LBP at intake with further episodes of mild LBP. Risk factors for developing or sustaining LBP were calculated using logistic regression analysis.

A more ankle-steered proprioceptive postural control strategy in upright standing increases the risk for developing or having recurrences of mild LBP within two years (Odds:1,165; 95% CI: 1,038 – 1,308; $p < 0.05$). Decreased postural robustness, altered spinal postural angles, psychosocial and physical activity outcomes were not identified as risk factors for future mild LBP.

Key words: proprioception, posture, postural control, prospective study, low back pain

1. Introduction

Non-specific low back pain (LBP) is one of the most frequent musculoskeletal disorders with high rates of reoccurrences. Life time prevalence is very high and 11-12 % of the people with LBP is disabled (Balague et al., 2012). As a result LBP is a major health problem for the western society resulting in high economical costs for the society (Carragee et al., 2005). Recently, studies on the causes and mechanisms for LBP were identified as a top primary care research priority for LBP research (Costa et al., 2013). Therefore, more research into risk factors for developing LBP by means of prospective studies needs further consideration.

Altered proprioceptive postural control (e.g. decreased use of lumbar proprioceptive inputs, more ankle-steered postural strategy) has frequently been shown in people with LBP (Brumagne et al., 2004; Claeys et al., 2011; della Volpe et al., 2006). However, a cause-effect relationship remains unclear because most studies were cross-sectional in design.

Indeed, until now few prospective studies investigated the cause-effect relation between altered postural control and the development of LBP. Increased posterior pelvic tilt and larger lumbar repositioning errors during sitting were shown to increase the risk for developing LBP in nursing students (Mitchell et al., 2010). Moreover, delayed trunk muscle latencies during sitting contributed to the development of LBP in college athletes (Cholewicki et al., 2005). Both prospective studies suggested proprioceptive deficits as an underlying mechanism, but a more direct evaluation of the proprioceptive system was not performed in these studies.

Besides postural control, also psychosocial variables were demonstrated to play a role in the development of LBP (Vlaeyen et al., 1995). Future serious LBP was strongly predicted by the baseline psychosocial characteristics (i.e. fear and distress) in different working populations (Carragee et al., 2005; Hiebert et al., 2012). Moreover, in a systematic review, depression, psychological distress, passive coping and fear avoidance beliefs were demonstrated to contribute in the transition from acute to chronic LBP (Ramond et al., 2011). However, the role of psychosocial variables in combination with proprioceptive postural control characteristics as predictors for LBP episodes remains unclear.

In addition to the psychosocial influences, also the role of physical activity remains obscure and ambiguous in the development of LBP (Heneweer et al., 2011). An U-shaped relation stated that people with moderate physical activity scores are less at risk for developing LBP compared to both people with extremely high or low physical activity scores (Heneweer et al., 2009). However, studies evaluating physical activity as a risk factor did not include postural control characteristics.

The aim of this prospective study was to investigate the role of proprioceptive postural control characteristics, psychosocial variables and physical activity in the development and/or maintenance of LBP in a young population. Baseline measurements were performed for proprioceptive steering, postural robustness, usual standing and usual sitting posture, pain, disability, physical activity and psychosocial characteristics. The LBP status was registered during a two year follow-up using questionnaires evaluating pain and disability.

2. **Materials and methods**

Subjects

A young population of 104 students participated voluntarily in this study. Test procedures were approved by the Medical Research Ethics Committee of KU Leuven with respect to the declaration of Helsinki (Ethical Principles for Medical Research Involving Human Subjects). All subjects gave their written informed consent. Participants were followed up during two years and the incidence of LBP was registered every three months by filling out the Oswestry Disability Index (ODI-2) (Fairbank and Pynsent, 2000) and by rating their back pain on a numeric rating scale (NRS) (Joos et al., 1991). During the follow-up period, subjects had to fill out four other questionnaires, every three months: A physical activity index (PAI) (Baecke et al., 1982), the Fear-Aavoidance Beliefs Questionnaire (FABQ) (Waddell et al., 1993), the Four-Dimensional Symptom Questionnaire (4DSQ) (Terluin, 1998) and the Tampa Scale of Kinesiophobia (TSK) (Roelofs et al., 2004). For all questionnaires, subjects were asked to rate their average status during the last month. Table 1 gives an overview of the characteristics of the subjects at intake. During the follow-up subjects were declared as having no LBP if the ODI-2 score was less than six and the NRS score was zero (Claeys et al., 2011). If one of both scores as higher the subjects were classified as having LBP. Ninety subjects completed the prospective study. Fourteen subjects decided to leave the study because they experienced filling out the questionnaires as too time-consuming. Figure 1 shows an overview of the subjects with the drop-outs.

Table 1. Characteristics of the subjects at baseline

Variable	Subjects with LBP Mean + SD	Subjects without LBP Mean + SD	Drop-outs	Significance
N	43 (10 M, 33 F)	61 (12 M, 49 F)	14 (4 M, 10 F)	
Age (years)	19.1 ± 1.6	19.2 ± 3.7	18.9 ± 2.7	NS
Height (cm)	174.0 ± 8.1	170.4 ± 7.4	174.4 ± 9.7	S
Weight (kg)	65.4 ± 8.8	63.3 ± 7.6	65.1 ± 9.7	NS
BMI	21.5 ± 2.2	21.8 ± 2.4	21.5 ± 3.5	NS
ODI-2	7.0 ± 4.5	1.8 ± 2.3	3.2 ± 2.6	S
NRS	2.8 ± 2.2	0	0.7 ± 1.3	S

LBP = non-specific low back pain, SD = standard deviation, N = number of subjects, M = male, F = Female, NS = no significant difference (p>0.05), S = significant difference (p<0.05), cm = centimeters, kg = kilogram, BMI = body mass index, ODI-2 = 0swestry disability index, NRS = numeric rating scale for pain

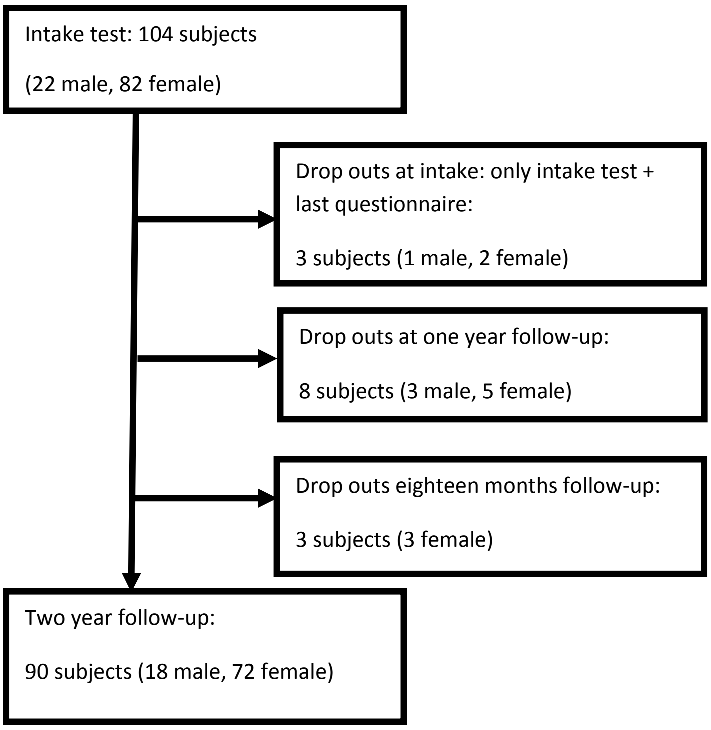


Fig. 1. Flowchart of the subjects participating in the study and drop-outs.

Postural balance analysis

Postural sway characteristics were measured using a six channel strain gauges force plate (Bertec Corporation, Ohio, USA). Force plate data were sampled at 500 Hz using a micro 1401 data-acquisition system and spike2 software (Cambridge Electronic Design, UK) and low pass filtered with a cutoff frequency of five Hz.

Muscle vibration

Muscle vibration was used to examine the role of proprioception in postural control. Muscle vibration stimulates muscle spindles and creates a lengthening illusion of the muscles (Roll and Vedel, 1982; Vedel and Roll, 1982). Two muscle vibrators (self-manufactured with Maxon motors, Switzerland) were used. Vibration was applied bilaterally to triceps surae muscles or to lumbar multifidus muscles, respectively. These muscles represent the muscles used in an ankle-steered strategy or a multi-segmental strategy (Brumagne et al., 2008). Muscle vibration was initiated 15 s after the start of the trial for the duration of 15 s. Activation and deactivation of the vibrators were manually controlled. The frequency of the vibration was set at 60 Hz and the amplitude was approximately 0.5 mm. These characteristics of vibration were demonstrated to induce a significant muscle lengthening illusion in healthy individuals (Cordo et al., 2005). Larger directional sways indicate that the central nervous system is using signals of the vibrated muscle. To avoid falling corrective displacements are made to compensate for the kinaesthetic illusions. For instance, vibration of triceps surae muscles in healthy subjects during standing can give the illusion of forward leaning and therefore the individual will compensate with a postural sway in a backward direction (Brumagne et al., 2004). When vibration is applied to lumbar multifidus muscles during standing a postural sway in a forward direction is expected (Brumagne et al., 2008).

Test procedure

A prospective study was used to investigate if proprioceptive postural control characteristics could be identified as risk factors for developing or having recurrences of LBP. To examine postural robustness and proprioceptive postural control, three postural positions were analyzed on the force plate: quiet standing on a stable support surface, quiet standing on an unstable support surface (foam) and sitting on a stool with stool and feet on the force plate, respectively (Claeys et al., 2011). The foam condition should force the subjects to rely less on ankle proprioceptive inputs and to change from an ankle-steered strategy to a multi-segmental strategy (Brumagne et al., 2008; Claeys et al., 2011). In the two standing conditions the subjects had to perform four trials: quiet standing, quiet standing with ballistic arm flexion, quiet standing with ankle muscle vibration and quiet standing with back muscle vibration. The subjects had to stand barefoot with the arms hanging relaxed along the body. During sitting subjects had to do three trials: usual sitting, usual sitting with ankle muscle vibration and usual sitting with back muscle vibration. The sitting condition was chosen as a condition in which subjects must rely more on back muscle proprioceptive inputs instead of ankle proprioceptive inputs to control postural balance (Claeys et al., 2011). The feet position (both heels 10 cm separated, both forefeet in a free splayed out position) was marked on a transparency sheet for standardization throughout the measurements (standing and sitting). In all postural balance trials, vision was occluded by means of non-transparent goggles. The subjects had to keep their eyes open, keeping their gaze in a straight-ahead direction. In all trials the subjects were asked to stand or sit in their usual standing or sitting position as immobile, but relaxed as possible. These experimental trials were carried out at intake. Fig. 2 shows the experimental setup of the proprioceptive postural control tests on the force plate during standing and sitting. After the postural balance tests, postural angles in usual standing and sitting were evaluated with digital photographs. Questionnaires were filled out after the experimental trials. These evaluations by questionnaires were repeated every three months during two years. Table 2 gives an overview of the proprioceptive postural control tests on the force plate.

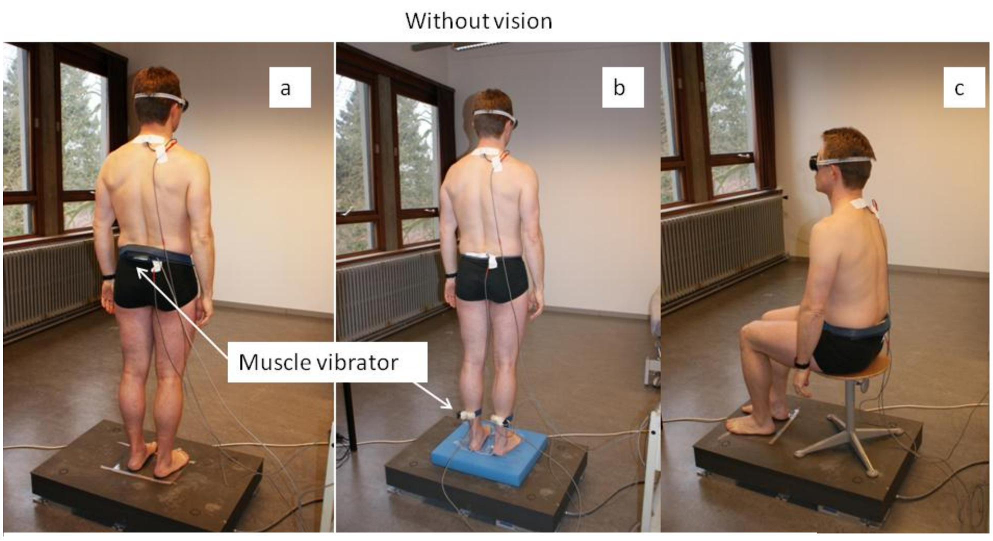


Fig. 2. Experimental set-up to investigate proprioceptive postural control:
a) standing on stable support surface with vibration on multifidus muscles;
b) standing on foam pad with vibration on soleus muscles;
c) sitting.

Table 2. Overview of the experimental trials to evaluate postural stability and proprioceptive postural control

Posture: Quiet standing	
Condition 1: stable support surface	
Trial 1	Quiet standing
Trial 2	Quiet standing, ballistic shoulder flexion to 90° at 30s
Trial 3	Quiet standing, bilateral triceps surae vibration
Trial 4	Quiet standing, bilateral lumbar multifidus muscle vibration
Condition 2: unstable support surface (foam)	
Trial 5	Quiet standing
Trial 6	Quiet standing, ballistic shoulder flexion to 90° at 30s
Trial 7	Quiet standing, bilateral triceps surae vibration
Trial 8	Quiet standing, bilateral lumbar multifidus muscle vibration
Posture: sitting on stool	
Trial 9	Sitting
Trial 10	Sitting, bilateral triceps surae vibration
Trial 11	Sitting, bilateral lumbar multifidus muscle vibration

Posture analysis

To assess the spinal posture in the sagittal plane, an experienced physiotherapist positioned manually photo-reflective markers on the six anatomical landmarks of the subjects using double-sided tape as follows: spinous process of cervical vertebra C7, spinous process of thoracic vertebra T12, spinous process of lumbar vertebra L3, spinous process of the sacrum S2, anterior superior iliac spine (right side), midpoint of the greater trochanter (right side). The spine was evaluated in the sagittal plane from the right side during usual standing and usual sitting. Five sagittal spinal angles were evaluated: pelvic tilt, lumbar curve, lumbar angle, thoracic flexion and trunk angle (O'sullivan et al., 2002). Subjects were asked to keep their gaze forward during the photographic assessment. To measure the angles, 2-D lateral photographs were taken with a digital photo camera (Sony A200 DSLR-A200K with lens DT18-70mm F3.5-5.6.), stabilized on a tripod with a height of 946mm and 6m away from the subject to standardize the tests. The digital photographs were imported in an image-processing program (MatLab R2008a, MathWorks Inc., Massachusetts USA) to determine the 2D-co-ordinates (X- and Y-co-ordinates) of each bony landmark. Nine postural angles were calculated by MathCAD 14 (PTC, USA) using formulas of trigonometry. The use of these markers in combination with digital photographs has been demonstrated to be a reliable method for postural research (Black 1996). The postural angles evaluated are illustrated in Fig. 3.

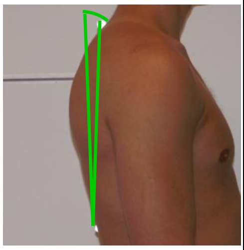
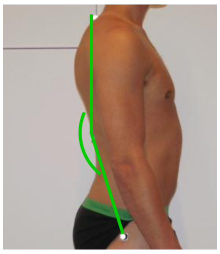
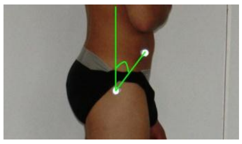
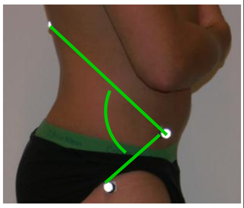
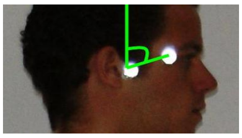
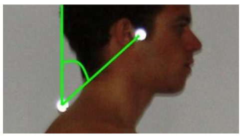
Lumbar curve (T12–L3–S2) 	Thoracic inclination (C7–T12–Vertical) 	Trunk angle (C7–T1–Trochanter) 	Pelvic tilt (ASIS–Trochanter– Vertical) 
Lumbar angle (T12–ASIS– Trochanter) 	Head angle (Eye–Ear–Vertical) 	Neck angle (Ear–C7–Vertical) 	Cervicothoracic angle (Ear–C7–T12 

Fig. 1. The postural angles

Data reduction and statistical analysis

Postural sway characteristics from the force plate readings were collected and calculated using Spike 2 (CED, Cambridge, UK) and Microsoft Excel software. Displacements of the center of pressure (COP) in anterior-posterior direction were estimated from the raw force data using the equation: COP= Mx/Fz. Root mean square (RMS) values of the COP displacements were calculated for postural robustness measures and mean values for the trials with muscle vibration to analyze the directional effect of muscle vibration on COP displacement. During the muscle vibration trials the COP displacements were analyzed over two periods: the 15 s preceding and the 15 s during muscle vibration. Forward COP displacement will give a positive value. Negative values correspond to backward COP displacement. To appraise the proprioceptive postural control strategy the following equation was used: RPW TS/LM= (absolute TS)/(absolute TS + absolute LM). In this equation RPW refers to relative proprioceptive weighting, absolute TS is the absolute value of the mean COP displacement during triceps surae muscle vibration and absolute LM is the absolute value of the mean COP displacement during lumbar multifidus muscle vibration. A RPW outcome of 1 corresponds to 100% reliance on ankle muscles afference. A RPW score of 0 indicates a 100% reliance on lumbar multifidus muscle afference.

Four different subgroups were determined after two years based on the NRS-pain en ODI-2 scores described above: Group 1 consisted of people with no LBP both at intake and during the two year follow-up (NoLBP-NoLBP), Group 2 consisted of people with no LBP at intake and who develop minimum one episode of LBP during the follow-up (NoLBP-LBP), Group 3 consisted of people with LBP at intake but no further episode of LBP (LBP-NoLBP), Group 4 consisted of people with LBP at intake and minimum one episode of LBP during the follow-up period (LBP-LBP).

Group differences of the RMS and mean values of the COP, the RPW values, postural angles, PAI and psychosocial factors were analyzed using a one-way ANOVA test. Where a significant main and interaction effect was found post hoc tests (Tukey's unequal N HSD) were performed to further analyze the detailed effects. All data are presented as mean $\pm$ standard deviation (SD). The level of statistical significance was set at $P< 0.05$. To determine the likelihood of developing or sustaining LBP after the intake test, logistic regression analysis was performed for the variables with statistically significant difference in the four group analysis. Next, a separate logistic regression analysis was performed with the significant variables (N= 2) of the first regression analysis. The outcome variable of the logistic regression analysis was at least one episode of LBP after the intake test or no future episode of LBP after the intake test. The variable US MV soleus was transformed into centimeters for the regression analysis to make the odds ratios more representative. Odds ratios and 95% confidence intervals (CI) were calculated. The statistical analysis was performed with SPSS Statistics 20 (IBM, USA).

3. Results

Characteristics of the subjects

A significant difference on the 4DSQ Fear scale existed within the NoLBP group at intake: the NoLBP-LBP group had larger scores on the 4DSQ Fear scale compared to the NoLBP-NoLBP group ($p < 0.05$). The LBP-NoLBP group had significantly larger scores on the FABQ physical activity (FABQ-PA) scale compared to both the NoLBP-NoLBP and the NoLBP-LBP group ($p < 0.05$). The LBP-LBP group scored higher than the NoLBP-LBP group on the FABQ-PA scale ($p < 0.05$). Subject characteristics are shown in Table 3.

Table 3. Characteristics of the four groups at baseline

	No LBP – No LBP		No LBP - LBP		LBP - No LBP		LBP - LBP		
	n = 22 (M = 7, F = 15)		**n = 30 (M = 3, F = 27)**		**n = 9 (M = 2, F = 7)**		**n = 29 (M = 6, F = 23)**		
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Significance
Age	20.5	3.8	20.5	2.0	21.0	1.9	19.9	0.9	NS
Height	172.5	6.3	**168.7**	**7.4**	**176.0**	**10.6**	172.5	6.8	**S**
Weight	62.3	6.5	63.8	8.1	65.7	11.8	64.9	7.4	NS
BMI	20.9	1.8	22.4	2.3	21.1	2.4	21.8	2.0	NS
ODI-2	1.0	1.4	2.3	2.8	6.8	3.6	7.6	4.8	**S**
NRS LBP	0.0	0.0	0.0	0.0	3.6	2.2	2.7	2.3	**S**
4DSQ Distress	4.4	3.3	7.0	6.6	5.2	3.9	8.3	5.0	NS
4DSQ Depression	0.1	0.2	0.6	1.0	0.0	0.0	0.3	0.8	NS
4DSQ Fear	**0.6**	**1.2**	**2.9**	**4.4**	1.8	1.2	2.0	2.1	S
4DSQ Somatisation	4.2	3.6	5.7	4.7	4.6	4.4	6.6	4.2	NS
FABQ Physical activity	**6.9**	**5.7**	**4.3**	**4.5**	**10.1**	**5.4**	**9.4**	**5.0**	**S**
FABQ Work	2.2	5.9	2.6	5.2	4.7	8.5	5.3	6.7	NS
TSK	32.2	4.8	32.0	4.8	34.8	4.4	34.4	5.4	NS
PAI Work	2.1	0.4	2.1	0.4	2.0	0.3	1.9	0.5	NS
PAI Sports	3.3	0.6	**3.4**	**0.5**	**2.6**	**1.2**	3.1	0.6	**S**
PAI Leisure Time	2.8	0.4	**3.1**	**0.6**	**2.4**	**1.0**	2.8	0.5	**S**
PAI Total Score	7.7	2.0	**8.5**	**0.8**	**7.1**	**2.1**	7.8	1.0	**S**

LBP = non-specific low back pain, n = number, SD = standard deviation, S = statistical significant difference ($p<0.05$), NS = non- statistical significant difference ($p>0.05$), BMI = Body Mass Index, NRS = Numeric Rating Scale for pain, 4 DSQ = Four-dimensional symptom questionnaire, FABQ = Fear Avoidance Beliefs Questionnaire, TSK = Tampa Scale for Kinesiophobia, PAI = Physical Activity Index (Baecke)

Postural robustness, proprioceptive steering and relative proprioceptive weighting

First, no significant differences between the four groups were demonstrated concerning postural robustness. Table 4 shows the postural robustness scores. Second, a significant difference in proprioceptive steering was identified at intake: the NoLBP-LBP group relied more on ankle proprioceptive inputs in the stable standing condition compared to the NoLBP-NoLBP group ($p< 0.05$). Table 5 shows the results of the proprioceptive steering trials. Third, significant differences in RPW were demonstrated: during standing on an unstable support surface, the NoLBP-LBP group showed significantly higher RPW-values compared to NoLBP-No LBP group ($p< 0.05$). In addition, in the sitting condition, the LBP-NoLBP showed significantly lower RPW values compared to the LBP-LBP group ($p < 0.05$). Figure 4 shows the RPW-scores of the different groups.

Table 4. RMS values of the center of pressure (m.) of the postural robustness trials at baseline

	No LBP – No LBP n = 22		**No LBP - LBP n = 30**		**LBP - No LBP n = 9**		**LBP - LBP n = 29**		
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Significance
RMS US	0.014	0.005	0.013	0.004	0.017	0.003	0.013	0.004	NS
RMS US with ballistic arm flexion	0.015	0.006	0.015	0.004	0.019	0.007	0.015	0.004	NS
RMS US foam	0.035	0.007	0.034	0.007	0.037	0.008	0.033	0.007	NS
RMS US with ballistic arm flexion foam	0.031	0.005	0.031	0.006	0.034	0.007	0.030	0.007	NS

LBP = non-specific low back pain, n = number, SD = standard deviation, NS = no significant difference ($p>0.05$), RMS = root mean square of the center of pressure, US = usual standing.

Table 5. Mean values of the center of pressure displacement during the muscle vibration trials at baseline (m).

	No LBP – No LBP n = 22		**No LBP - LBP n = 30**		**LBP – No LBP n = 9**		**LBP - LBP n = 29**		
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Significance
US MV soleus	**-0.085**	**0.051**	**-0.121**	**0.043**	-0.112	0.036	-0.106	0.046	**S**
US MV multifidus	0.027	0.019	0.036	0.021	0.045	0.036	0.031	0.025	NS
US MV soleus foam	-0.039	0.022	-0.057	0.035	-0.041	0.017	-0.050	0.030	NS
US MV multifidus foam	0.050	0.025	0.045	0.028	0.042	0.021	0.046	0.028	NS
USit MV soleus	0.001	0.002	0.001	0.004	0.002	0.004	0.000	0.003	NS
USit MV multifidus	-0.016	0.009	-0.017	0.010	-0.018	0.005	-0.016	0.012	NS

LBP = non-specific low back pain, n = number, SD = standard deviation, S = Significant difference ($p<0.05$), NS = no significant difference ($p>0.05$), RMS = root mean square of the center of pressure, US = usual standing, Usit = usual sitting, MV = muscle vibration.

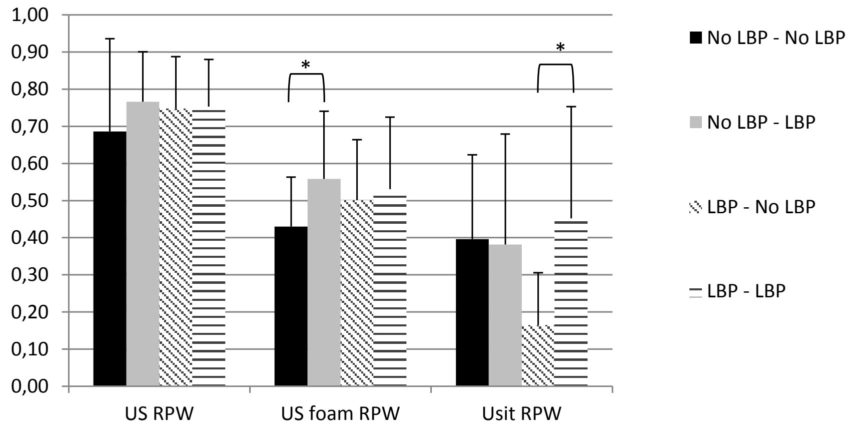


Fig. 4. Relative proprioceptive weighting scores of the subjects at intake.

Postural angles

No significant differences in posture could be identified between the different groups (P> 0.05). Table 6 shows the results of the postural angles during usual standing and sitting.

Table 6. Postural angles at baseline in degrees.

	No LBP – No LBP n = 22		No LBP - LBP n = 30		LBP - No LBP n = 9		LBP - LBP n = 29		
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Significance
Lumbar Curve US	155.6	18.2	149.7	15.0	156.8	5.8	156.5	9.5	NS
Thoracic Flexion US	8.2	15.5	7.0	11.9	3.7	1.9	6.9	11.5	NS
Trunk Angle US	202.5	5.2	206.9	6.0	202.4	8.4	209.7	17.4	NS
Pelvic Tilt US	37.0	9.4	38.7	7.9	38.4	3.3	35.6	12.2	NS
Lumbar Angle USit	97.3	13.0	92.7	15.5	94.4	3.8	95.6	18.4	NS
Lumbar Curve Usit	170.6	13.5	165.5	36.4	167.8	19.3	172.3	5.3	NS
Thoracic Flexion Usit	16.2	16.5	15.6	25.0	15.3	13.4	14.4	11.5	NS
Trunk Angle Usit	225.5	5.8	228.5	4.8	222.5	20.4	225.8	14.0	NS
Pelvic Tilt Usit	20.9	15.8	18.9	13.7	18.2	22.2	20.0	13.5	NS
Lumbar AngleUSit	125.2	13.2	119.5	10.1	123.0	13.4	113.4	31.9	NS

LBP = non-specific low back pain, n = number, SD = standard deviation, NS = no significant difference, US = usual standing, Usit = usual sitting

Predictors of future LBP

The logistic regression model contained the nine variables with statistically significant difference in the four group analysis. Next, the regression analysis is repeated with the two significant predictors. In the final model, two significant predictors were more sway during soleus muscle vibration in stable standing ($P< 0.05$; Odds 1.165; 95% CI: 1.038 – 1.308) and 4DSQ Fear ($P< 0.05$; Odds 0.708; 95% CI: 0.521 – 0.963). Results are shown in Table 7.

Table 7. Logistic regression predicting LBP during the follow-up.

Regression analysis with the nine significant variables								
	B	**S.E.**	**Wald**	**df**	**Sig.**	**Odds Ratio**	**95% C.I. for Odds Ratio**	
							Lower	**Upper**
Height	0.079	,043	3,381	1	,066	1,082	,995	1,178
PAI Sports	-1,176	,815	2,081	1	,149	,309	,062	1,524
PAI Leisure Time	-1,522	,857	3,157	1	,076	,218	,041	1,170
PAI Total Score	,871	,640	1,855	1	,173	2,389	,682	8,369
4DSQ Fear	**-,559**	**,213**	**6,919**	**1**	**,009**	**,572**	**,377**	**,867**
FABQ Physical activity	,069	,056	1,521	1	,218	1,071	,960	1,195
US MV soleus	**,209**	**,079**	**6,988**	**1**	**,008**	**1,233**	**1,056**	**1,439**
US foam RPW	-2,730	1,847	2,186	1	,139	,065	,002	2,433
Usit RPW	-1,536	1,151	1,781	1	,182	,215	,023	2,055
Final model								
4DSQ Fear	**-,345**	**,157**	**4,843**	**1**	**,028**	**,708**	**,521**	**,963**
US MV soleus	**,153**	**,059**	**6,749**	**1**	**,009**	**1,165**	**1,038**	**1,308**

PAI = Physical Activity Index, 4DSQ = four-dimensional symptom questionnaire, FABQ = fear avoidance beliefs questionnaire, US = usual standing, MV = muscle vibration, RPW = relative proprioceptive weighting, Usit = usual sitting.

4. Discussion

The main finding of this study is that a more ankle-steered proprioceptive postural control strategy during stable standing slightly increases the risk for developing or sustaining mild LBP within two years. In contrast, decreased postural robustness, postural differences in usual standing and sitting, psychological variables and physical activity level were not demonstrated as risk factors in this student population. Remarkably, higher scores on the 4DSQ Fear scale reduce the risk for developing or maintaining LBP.

No differences in postural robustness between the four prospective groups at intake were observed, which is in contrast with previous studies (Table 4) (Brumagne et al., 2008; della Volpe et al., 2006; Mok et al., 2004). The low pain and disability scores of the population studied in this study may clarify these findings. Subjects were classified as having LBP if the NRS-pain score was more than zero or when disability on the ODI-2 was more than six percent. Consequently, the pain and disability scores of the patients may be clinically too mild to alter postural robustness. Previous studies demonstrating decreased postural robustness in people with LBP consisted of patients with higher pain and/or disability scores (della Volpe et al., 2006; Mok et al., 2004; Popa et al., 2007). Moreover, the postural tasks used in the current study might not have been challenging enough to appraise postural robustness differences between groups.

Although no differences were found in postural robustness between the four groups, some noteworthy proprioceptive postural control differences need further attention. The NoLBP-LBP group showed higher reliance on ankle muscle proprioceptive signals during soleus vibration in standing on a stable surface compared to the NoLBP-NoLBP group (Table 5). Moreover, during standing on an unstable surface higher RPW values (i.e. more ankle-steered control strategy) were demonstrated in the NoLBP-LBP group compared to the NoLBP-NoLBP group. The NoLBP-NoLBP group did not show an increased reliance on ankle proprioceptive signals for standing postural control compared to those who developed mild LBP within two years. This is in agreement with the results of previous cross-sectional studies where a decreased reliance on back muscle proprioceptive inputs for standing postural control was demonstrated (Brumagne et al., 2008).

In contrast to these cross-sectional studies, the current study was a prospective analysis and identified differences at baseline within the proprioceptive system between healthy people developing LBP and healthy people remaining healthy. Indeed, people with a clear ankle-steered proprioceptive postural control strategy in stable standing showed slightly higher odds to develop LBP in the future. This indicates that in this young population with mild LBP, the LBP may be caused by an altered proprioceptive reweighting and that the observed changes in proprioceptive postural control are not only the result of LBP as frequently suggested in earlier cross-sectional studies (Brumagne et al., 2008; della Volpe et al., 2006). High reliance on ankle muscle proprioceptive inputs and the concomitant rigid proprioceptive postural control strategy in people who develop LBP may result in a less fine-tuned control of the spine during postural tasks. Hence, the mechanical stress on the lumbar spine may increase which could lead to spinal injury and pain (Cholewicki et al., 2005).

The LBP-NoLBP group showed significantly lower RPW values during sitting compared to the LBP-LBP group (Fig. 4). This indicates that people with LBP who use more back muscle proprioceptive signals for sitting postural control are more likely to become healthy in the near future (two years) compared to those who use less back muscle proprioceptive afference during sitting. However, it must be noticed that the LBP-NoLBP group is very small (nine subjects) compared to the other groups. As a result caution should be exercised when interpreting these results.

Despite the clearly demonstrated proprioceptive postural control differences during standing and sitting, postural differences were not demonstrated between groups in the current study (Table 6). These findings are in agreement with previous studies which could not demonstrate postural differences in usual standing and sitting between people who develop LBP and healthy controls (Mitchell et al., 2010; Mitchell et al., 2009). Symptoms in the current study may be too mild to be associated with postural differences between groups. Moreover, the studies that have been demonstrating postural differences subclassified their patients based on aggravating movements and postures (Dankaerts et al., 2006).

Fear and fear-avoidance beliefs may be ruled out as a risk factor in the development of mild LBP in this young population based on the current results. Patients with scores lower than 14 on the FABQ-PA were not demonstrated to be significantly more at risk for developing LBP in the first six months (George et al., 2008). Accordingly, the fairly low fear-avoidance beliefs in the current study may not result in future LBP, because the FABQ-PA scores were not higher than 10.1. Finally, it's remarkable that more fear may reduce the risk for developing or maintaining LBP based on the 4DSQ fear questionnaire. However, the minimal important clinical threshold for fear on this questionnaire is 8 (range 0 – 24) and the highest mean score in one test group was not above 3. Therefore, caution should be exercised when fear is considered as a contributing factor to the development of LBP. Altogether, an altered proprioceptive control as an underlying mechanism in the development of mild LBP in this young population becomes more explicit. Despite the evaluation of psychosocial, physical activity level, postural and postural robustness variables, only an ankle-steered proprioceptive control strategy could be identified as a clear risk factor for developing LBP during two year follow-up in this young student population. Moreover, the level of LBP in the current study was very mild and these mild symptoms could be predicted by evaluating the prospective system at baseline. These findings may further emphasize the role of the proprioceptive system as an underlying mechanism.

To our knowledge, the current prospective study is the first study revealing proprioceptive deficits during a postural control task that was associated with the development of mild LBP in the near future. Moreover, changes in the proprioceptive system were specifically evaluated by means of muscle vibration and not hypothesized as in most other studies (Mok et al., 2007; Popa et al., 2007).

As a result, some conclusions can be drawn about the rehabilitation and prevention of LBP. Motor output and postures are readily available to both, clinicians and researchers. Therefore, we are often inclined to direct our examination and treatment solely on motor output and postures. The sensory input and processing of this afference, which may lead to changes in motor output and postures, are often neglected. However, the results of this study suggest that changes in proprioceptive processing may already occur without obvious changes in motor output (e.g. postural robustness) and postures, but increase the risk for mild LBP. Possibly, the underlying mechanism (altered proprioception) must be present long enough to result in visible motor output changes. Despite these authors concur with the central role of optimizing posture in the rehabilitation of patients with LBP, addressing the sensory component, and not only the motor part, may proof fruitful in the prevention and rehabilitation of LBP.

5. Limitations and future directions

In spite of the demonstrated proprioceptive difference during an intake test between people who develop mild LBP compared to healthy controls, the total number of subjects may be an important limitation of the current study. Only 90 subjects completed the prospective two year follow-up. One of the subgroups (LBP-NoLBP) consisted of nine subjects only. It must be noticed that equal group size may result in more statistically significant differences such as the RPW values in the regression analysis. Therefore, larger prospective studies are necessary to further underpin the novel findings of this study.

Identifying risk factors may be crucial to reduce the high frequency of LBP. A more ankle-steered proprioceptive postural control strategy as a potential risk factor was identified in a laboratory setting by means of muscle vibration. However, this evaluation method is not entirely feasible in a clinical setting. Developing tests to identify proprioceptive steering in a more clinical setting may be a crucial step in the evaluation, prevention and more optimal rehabilitation of LBP (Brumagne et al., 2013)

Despite the specific evaluation of the proprioceptive system by means of muscle vibration, it remains unclear if these proprioceptive control changes are based on changed peripheral inputs (at muscle spindle level) or changed sensory processing (e.g. reweighting, at brain level) or a combination of both. Future research using muscle vibration in combination with brain imaging (e.g. fMRI, NIRS) during postural control tasks may help to clarify this research question.

6. Conclusion

Increased reliance on ankle muscle proprioceptive inputs during standing on a stable surface slightly increases the risk for future mild LBP in young individuals. In contrast, postural robustness, postural angles, psychosocial variables and physical activity level were not associated with the development or recurrences of LBP in this student population. Therefore, addressing proprioceptive input and processing impairments may proof fruitful in the prevention and rehabilitation of LBP.

7. Acknowledgements

Lotte Janssens received a Ph.D. fellowship from the Research Foundation Flanders (FWO).

8. References

1. Baecke JA, Burema J, Frijters JE. A short questionnaire for the measurement of habitual physical activity in epidemiological studies. Am. J. Clin. Nutr. 1982;36(5):936-942.
2. Balague F, Mannion AF, Pellise F, Cedraschi C. Non-specific low back pain. Lancet 2012;379(9814):482-491.
3. Black KM, McClure P, Polansky M. The influence of different sitting positions on cervical and lumbar posture. Spine1996;21(1):65-70.
4. Brumagne S, Cordo P, Verschueren S. Proprioceptive weighting changes in persons with low back pain and elderly persons during upright standing. Neurosci. Lett. 2004;366(1):63-66.
5. Brumagne, S, Janssens L, Claeys K, and Pijnenburg M. Chapter 12: Altered variability in proprioceptive postural strategy in people with recurrent low back pain and healthy individuals. In:Hodges, PW, Cholewicki J., Van Dieën J. (Eds),Spinal Control: The Rehabilitation of Back Pain - State of the Art and Science. 2013;Elsevier Churchil Livingstone, Edinburgh, p. 135-144.ISBN:978-0-7020-4356-7.
6. Brumagne S, Janssens L, Knapen S, Claeys K, Suuden-Johanson E. Persons with recurrent low back pain exhibit a rigid postural control strategy. Eur. Spine J. 2008;17(9):1177-1184.
7. Carragee EJ, Alamin TF, Miller JL, Carragee JM. Discographic, MRI and psychosocial determinants of low back pain disability and remission: a prospective study in subjects with benign persistent back pain. Spine J. 2005;5(1):24-35.
8. Cholewicki J, Silfies SP, Shah RA, Greene HS, Reeves NP, Alvi K, Goldberg B. Delayed trunk muscle reflex responses increase the risk of low back injuries. Spine 2005;30(23):2614-2620.
9. Claeys K, Brumagne S, Dankaerts W, Kiers H, Janssens L. Decreased variability in postural control strategies in young people with non-specific low back pain is associated with altered proprioceptive reweighting. Eur. J. App. Physiol. 2011;111(1):115-123.

10. Cordo PJ, Gurfinkel VS, Brumagne S, Flores-Vieira C. Effect of slow, small movement on the vibration-evoked kinesthetic illusion. Exp. Brain Res. 2005;167(3):324-334.
11. Costa LC, Koes BW, Pransky G, Borkan J, Maher CG, Smeets RJ. Primary care research priorities in low back pain: an update. Spine 2013;38(2):148-156.
12. Dankaerts W, O'Sullivan P, Burnett A, Straker L. Differences in sitting postures are associated with nonspecific chronic low back pain disorders when patients are subclassified. Spine 2006;31(6):698-704.
13. della Volpe R, Popa T, Ginanneschi F, Spidalieri R, Mazzocchio R, Rossi A. Changes in coordination of postural control during dynamic stance in chronic low back pain patients. Gait. Posture. 2006;24(3):349-355.
14. Fairbank JC, and Pynsent PB. The Oswestry Disability Index. Spine 2000;25(22):2940-2952.
15. George SZ, Fritz JM, Childs JD. Investigation of elevated fear-avoidance beliefs for patients with low back pain: a secondary analysis involving patients enrolled in physical therapy clinical trials. J. Orthop. Sports Phys. Ther. 2008;38(2):50-58.
16. Heneweer H, Staes F, Aufdemkampe G, van RM, Vanhees L. Physical activity and low back pain: a systematic review of recent literature. Eur. Spine J. 2011;20(6):826-845.
17. Heneweer H, Vanhees L, Picavet HS. Physical activity and low back pain: a U-shaped relation? Pain 2009;143(1-2):21-25.
18. Hiebert R, Campello MA, Weiser S, Ziemke GW, Fox BA, Nordin M. Predictors of short-term work-related disability among active duty US Navy personnel: a cohort study in patients with acute and subacute low back pain. Spine J. 2012;12(9):806-816.
19. Joos E, Peretz A, Beguin S, Famaey JP. Reliability and reproducibility of visual analogue scale and numeric rating scale for therapeutic evaluation of pain in rheumatic patients. J. Rheumatol. 1991;18(8):1269-1270.
20. Mitchell T, O'sullivan PB, Burnett A, Straker L, Smith A, Thornton J, Rudd CJ. Identification of modifiable personal factors that predict new-onset low back

pain: a prospective study of female nursing students. Clin. J. Pain 2010;26(4):275-283.

21. Mitchell T, O'sullivan PB, Smith A, Burnett AF, Straker L, Thornton J, Rudd CJ. Biopsychosocial factors are associated with low back pain in female nursing students: a cross-sectional study. Int. J. Nurs. Stud. 2009;46(5):678-688.
22. Mok NW, Brauer SG, Hodges PW. Hip strategy for balance control in quiet standing is reduced in people with low back pain. Spine 2004;29(6):E107-E112.
23. Mok NW, Brauer SG, Hodges PW. Failure to use movement in postural strategies leads to increased spinal displacement in low back pain. Spine 2007;32(19):E537-E543.
24. O'sullivan PB, Grahamslaw KM, Kendell M, Lapenskie SC, Moller NE, Richards KV. The effect of different standing and sitting postures on trunk muscle activity in a pain-free population. Spine 2002;27(11):1238-1244.
25. Popa T, Bonifazi M, Della VR, Rossi A, Mazzocchio R. Adaptive changes in postural strategy selection in chronic low back pain. Exp. Brain. Res. 2007;177(3):411-418.
26. Ramond A, Bouton C, Richard I, Roquelaure Y, Baufreton C, Legrand E, Huez JF. Psychosocial risk factors for chronic low back pain in primary care--a systematic review. Fam. Pract. 2011;28(1):12-21.
27. Roelofs J, Goubert L, Peters ML, Vlaeyen JW, Crombez G. The Tampa Scale for Kinesiophobia: further examination of psychometric properties in patients with chronic low back pain and fibromyalgia. Eur. J. Pain 2004;8(5):495-502.
28. Roll JP, and Vedel JP. Kinaesthetic role of muscle afferents in man, studied by tendon vibration and microneurography. Exp. Brain Res. 1982;47(2):177-190.
29. Terluin B. De Vierdimensionele Klachtenlijst (4DKL) in de huisarspraktijk - psychodiagnostisch gedreedschap. 33, 18-24. 1998.
30. Vedel JP, and Roll JP. Response to pressure and vibration of slowly adapting cutaneous mechanoreceptors in the human foot. Neurosci. Lett. 1982;34(3):289-294.
31. Vlaeyen JW, Kole-Snijders AM, Boeren RG, van EH. Fear of movement/(re)injury in chronic low back pain and its relation to behavioral performance. Pain 1995;62(3):363-372.

32. Waddell G, Newton M, Henderson I, Somerville D, Main CJ. A Fear-Avoidance Beliefs Questionnaire (FABQ) and the role of fear-avoidance beliefs in chronic low back pain and disability. Pain 1993;52(2):157-168.

Chapter 6

General discussion

1. Proprioceptive postural control and non-specific low back pain

The main finding of this doctoral thesis is that young people with mild LBP show an altered proprioceptive postural control compared to the healthy persons. They have a decreased variability in postural control strategies, an altered proprioceptive steering and decreased postural robustness in more difficult postural tasks (Study 1). The altered proprioceptive steering was also associated with a delayed preparatory pelvic control during the STSTS movement (Study 2) and could be identified as a risk factor for developing or sustaining LBP in the near future (Study 4). Usual postural positions were not associated with mild LBP in this study population and are not recognized as risk factor for developing or sustaining LBP (Study 4). Finally, the pelvis plays a crucial role in dynamic (STSTS movement) postural control, but less in static postural control (Study 2 and Study 3).

a. Proprioceptive impairment as a contributing underlying mechanism of non-specific low back pain

Changes in the proprioceptive system are often hypothesized as an important contributing underlying mechanism in people with LBP (della Volpe et al., 2006; Henry et al., 2006; Mok et al., 2004; Mok et al., 2007; Mok et al., 2011). In contrast to these studies, current doctoral thesis demonstrated proprioceptive changes using a specific evaluation of the proprioceptive signals. A decreased capacity to upweight multifidus proprioceptive afference is clearly observed in the three postural conditions (standing on a stable support surface, standing on an unstable support surface and sitting) in people with mild LBP (Study 1). This finding is particularly remarkable in the sitting condition, a condition where the back muscle (e.g. multifidus) signals are hypothesized to play a crucial role for optimal postural control (Brumagne et al., 1999; MacDonald et al., 2006). This decreased reliance on multifidus proprioceptive signals may result in a less fine-tuned segmental control of the lumbar spine during postural control. Suboptimal spinal control may result in higher mechanical stress on the lumbar spine, which may lead to more spinal pain (Cholewicki et al., 2005).

A subgroup of initially healthy people at intake, but developing LBP during the follow up, showed a similar reliance on ankle muscle proprioceptive inputs for standing postural control to people with LBP at intake. Consequently, this suggests that in a healthy population some individuals may already show proprioceptive characteristics, similar to that of people with LBP, without having pain or being disabled. However, based on the results of this doctoral thesis, these persons are almost four times more likely to develop LBP in the following years compared to individuals who rely on back muscle proprioceptive input for postural control (multi-segmental control strategy).

In addition, persons with a history of LBP are more at risk for developing future LBP (Cholewicki et al., 2005; Mitchell et al., 2010). The decreased proprioceptive reweighting capacity may also be a mechanism increasing the risk for future LBP episodes in subjects already having LBP. To our knowledge, this is the first study demonstrating specifically evaluated proprioceptive impairments as a risk factor for future LBP.

b. Postural control strategy and non-specific low back pain

A stronger ankle steered postural control strategy is observed in people with LBP compared to healthy controls. This conclusion is in agreement with earlier studies (Brumagne et al., 2004; Brumagne et al., 2010; Brumagne et al., 2008a; Brumagne et al., 2008b). Compared to these previous studies three important differences should be highlighted. First, the current study demonstrates this stronger ankle-steered postural control strategy specifically in a young population with very mild pain and disability scores. As a result pain is unlikely the causing factor in the selected postural strategy. Second, also a decreased variability in postural control strategies is observed in people with LBP. An ankle-steered postural control strategy could be sufficient in easy postural conditions, but may fail when the complexity of the postural task increases. The greater the variability in postural control strategies the better the opportunity is to select the optimal strategy upon the condition. This variability in postural control strategy may reduce the stress on the biomechanical system and may prevent tissue damage of the lumbar spine (Harbourne and Stergiou, 2009). Third, Study 4 is a prospective study and showed significantly higher odds for developing LBP in people using an ankle-steered postural control strategy during stable standing. Thus, a causal link between the use of an ankle-steered postural control strategy and the development of LBP may exist based on the results of the current doctoral thesis.

Decreased postural robustness has also been shown in people with LBP, especially when the task complexity increased (e.g. unstable standing with ballistic arm movement; Study 1). However, in contrast to the proprioceptive steering, decreased postural robustness could not be identified as a risk factor for developing LBP in this doctoral thesis. One may conclude that the performance of a postural task may be influenced by LBP. However, Study 4 demonstrated that in this population this altered performance did not increase the risk for developing LBP. This suggests that proprioceptive processing during postural control may be crucial in the development of LBP and not only the motor output of the postural task (Brumagne et al., 2008a; Brumagne et al., 2008b; Leinonen et al., 2003).

In contrast to the proprioceptive steering, postural angles in usual standing and sitting did not differ between people developing mild LBP and healthy controls (Study 4). This finding may be attributed to the low pain and disability scores in this young population. In addition, these results might also be caused by the fact that no formal sub-classification of subjects was applied. Previous studies have indeed highlighted the importance of sub-classifying people with LBP based on pain aggravating postures and movements (Dankaerts et al., 2006).

Interestingly, Study 3 showed some small to moderate correlations with the pelvis during usual sitting. During this posture the pelvis can be seen as a “steering wheel” dictating lumbar, thoracic and cervical postural angles. In clinical practice nowadays much attention is paid to ‘optimal’ sitting posture. Adequate pelvic control may form the base to instruct this ‘optimal’ sitting posture (Kuo et al., 2009; Leinonen et al., 2003). This pelvic bottom-up postural adjustment was less manifest during usual standing. In this position, also the trunk was shown to play an important role. During usual sitting, the mobile part of the body starts at the pelvis. In contrast, in usual standing also the joints of the lower limbs are part of the mobile part of the body. As a result, pelvic corrections only are likely insufficient to control the center of mass within the support base, which results also in spinal postural corrections at lumbar and thoracic level to keep optimal equilibrium.

In contrast to static positions, the role of the pelvis is more crucial in the transition from sitting to standing. Pelvic control is disturbed during STSTS in people with LBP (Study 2). Decreased use of lumbar proprioceptive signals during sitting and standing (evaluated with muscle vibration) is shown to be associated with a delay in preparatory pelvic control when moving from sitting to standing. This delay may not only slow down the performance, but may have some clinical consequences. A higher flexion moment of the lumbar spine may be the result of the delayed anterior pelvic movement onset observed in people with LBP. Consequently, this end-range flexion combined with high daily frequency may increase the risk for intervertebral disc injuries (Callaghan and McGill, 2001; Cholewicki and McGill, 1996; McGill, 2004).

Psychological variables are frequently demonstrated to be associated with current or future LBP (Carragee et al., 2005; Mitchell et al., 2010; Mitchell et al., 2009). However, based on the results of this doctoral thesis, they do not play a major role in young individuals with mild LBP under investigation in the current project. Scores on the TSK scale (healthy: 31.1$\pm$5.5 vs. LBP: 33.1$\pm$4.9) were not statistically different between people with and without LBP in Study 1. FABQ PA scores (healthy: 4.4$\pm$5.8 vs. LBP: 8.9$\pm$5.9) were statistically different between the study populations in Study 2, however, these scores did not reach the clinically important threshold (= 14) (George et al., 2008; Guclu et al., 2012). Moreover, regression analysis did not show higher odds for the fear and kinesiophobia scores for developing or sustaining LBP. These findings suggest that fear scores must reach a certain threshold to contribute to current or future LBP. Moreover, FABQ W scores are demonstrated to be more predictive for future LBP rather than FABQ PA scores (George et al., 2008). Subjects in the current study were all university students. They were instructed that studying must be interpreted as work when filling out the questionnaires. This may clarify the very low scores on the FABQ W questionnaires.

Physical activity scores of the subjects did not show significant differences between people with and without LBP in the cross-sectional analysis. Significant differences in physical activity were observed in the prospective study between the No LBP – LBP group and the LBP – No LBP group concerning PAI sports, PAI Leisure time en PAI total. However, caution is required to interpret higher scores on these PAI scale as a clear risk factor. A U-shape relation between physical activity and LBP illustrates higher risks for people with extremely low or high physical activity scores (Heneweer et al., 2009). Scores in Study 4 were moderate physical activity scores for all groups, despite a statistical difference between two out of the four groups. As a result, to our opinion and in accordance with the findings of Heneweer et al., these statistically different scores for physical activity may not be considered as a clear risk factor for developing LBP. Moreover, physical fitness rather than self-reported physical activity levels may be more predictive for future LBP (Heneweer et al., 2012).

2. Limitations and future directions

Some remarks concerning the test group need to be discussed. A young population with mild pain and disability scores was tested in the current project. Hence, results cannot be generalized to the general LBP population having different age, pain and disability scores. Moreover, a sub-classification based on pain aggravating postures and movements was not performed in the current study. Possibly, people have a robust postural control in pain-free postures but a less robust postural control in painful postures. Also the number of subjects per group may be a limitation in the current project, especially for the prospective analysis. Only 90 persons completed the prospective follow-up. One group consisted of only nine subjects. As a result, statistical power of the regression analysis may be decreased. Larger groups at baseline may be required to reduce the negative impact of drop-outs (Cholewicki et al., 2005). A last limitation about test group may be the unequal gender distribution between the groups. However, there may exist some gender differences concerning length, muscle mass, BMI and other anthropometric characteristics. Moreover, females are observed to report more frequently LBP and the duration of the pain episode is mostly longer in females (Leboeuf-Yde et al., 2009). In current project the ratio of females was much greater in all test groups (healthy and LBP) but this ratio is not always equal. The ratio female/male is larger in the patient groups of study 1 and study 2 and in the group developing LBP in study 4. These findings may indicate that gender may influence the results. However, to our knowledge, there is no evidence for gender differences concerning proprioceptive postural control in young people. Despite, the role of gender in proprioceptive postural control warrants further investigation in future.

A proprioceptive risk factor for developing or maintaining LBP was identified in the current thesis. However, it remains unclear if this risk factor can be modified. Therefore, intervention studies addressing these proprioceptive impairments, specifically in a group with a clear ankle-steered postural control strategy in stable standing, with a long follow-up period are required to further clarify this research question.

Pelvic kinematics were not included in Study 4 (Chapter 5) as possible developing variables for LBP. The main purpose of this study was to investigate the proprioceptive postural control variables in combination with the psychological variables and physical activity. Nevertheless, in future studies, evaluating also kinematic variables as risk factors in the development of LBP may be relevant based on the findings of Study 2 (Chapter 3)

Muscle vibration was used to investigate the proprioceptive steering. Skin thickness could influence the intensity of the vibration signal to the muscle spindles. The BMI of the subjects in these studies was under 25 and weight did not differ significantly between the groups. As a result, it may be hypothesized that differences in proprioceptive steering may not be caused by differences in skin thickness between the groups. In future studies, however, we advise to evaluate skinfold thickness in the areas where muscle vibration is applied, there, despite the low BMI values, regional fat distribution may differ between subjects and between genders (Coin et al., 2012; Kwok et al., 2011; Kwok et al., 2012; Yan et al., 2013). Evaluating the skinfolds over the vibrated muscles may be a more precise method to analyze the influence of regional fat distributions during the muscle vibration trials.

The determination of postural control strategies used by the subjects (ankle-steered vs. multisegmental steered) are based on the response to sensory stimulation of the muscle spindles of the postural muscles that are recognized as the main actors in these postural strategies (i.e. ankle muscles and back muscles). However, joint kinematics based on 3D-motion analysis may further underscore the conclusions about the postural control strategies selected by the subjects. Future research with movement analysis during the vibration trials may help to clarify the postural strategies of the subjects.

Despite the standardization of the frequency and the amplitude of the vibration signal, pressure may influence the vibration signal. However, the motor controller of the muscle vibrator adapts to the pressure and the muscle vibrators are attached on the strap and not between the strap and the body, which facilitates separate movement of the vibrator. Moreover, the method of application the muscle vibrators with non-elastic velcro was frequently exercised by the investigator to standardize through the different test sessions. As a result the influence of pressure on the vibration signal may be of less importance.

Displacements of the COP may be influenced by the height of the subjects. However, there was no significant difference in height between subjects in all studies (1, 2 and 4), except between the prospective groups 'No LBP – LBP' (study 4, table 3). But, these groups didn't show different RMS values of the COP (study 4, table 4). Nevertheless, correction of the COP excursions based on height and foot length may further optimize these results in future studies.

Two dimensional accelerometers, used to evaluate the kinematics of the STSTS, give only sagittal movement information. Rotational kinematics in the coronal plane were not evaluated. However, compensatory movements in the transversal plane during the STSTS may higher the load on spinal structures and may play a crucial role in the development of spinal pain (Shum et al., 2007). In addition, only the pelvic and upper thoracic spine kinematics were evaluated during the STSTS. Evaluating other body segments (knee, hip, lower lumbar spine, upper lumbar spine, lower thoracic spine) during the STSTS using 3-dimensional accelerometers or a 3-D motion analysis system may give additional information to better understand the changed kinematics in people with LBP. Finally, slower pelvic rotation onsets were only cross-sectionally demonstrated. Similar to the proprioceptive steering it may be fruitful to evaluate if delayed preparatory control of the pelvis may play a role in the development of LBP.

Questionnaires are used to evaluate fear-avoidance beliefs, both at the moment of the intake test and periodically during the two year follow-up of the prospective part of the study. However, actual fear at the moment of the postural control tests may also influence postural control. Actual fear could be evaluated by questionnaires but a more reliable method may be measuring skin conductance (Zeidan et al., 2012). But the objective in current project was to evaluate fear avoidance beliefs periodically in contrast to actual fear. Evaluating actual fear during the proprioceptive postural control test may be an important 'control' variable to include in future studies evaluating postural control.

Recall bias may also be an important limitation in research using questionnaires to evaluate pain and disability. Weekly recall may be as reliable as momentary electronic data (Jamison et al., 2006). Daily recall reduces patient satisfaction, which also influences the reliability of the data negatively (Gendreau et al., 2003). However, the study of Gendreau et al. (2003) shows that data collection in a familiar environment of the subjects is more reliable than that data collection in the laboratory. Moreover, data collection using electronic diaries increases patient satisfaction rather than data collection with paper diaries. Current study, having the limitation because of the quarterly data collection, used electronic questionnaires that people could be filled in at home (except at the intake test). People were informed that the study took 2 years, which in our opinion stimulated them to remember the possible episodes of pain and disability.

An important consideration is the use of the RMS-scores as measure for postural robustness. Less sway is interpreted as more robust. However, optimal robustness includes not only a stable behavior of the system, but also the possibility to explore during postural perturbations to achieve postural stability (Reeves et al., 2007). In our opinion, based on the findings of study 1, there is a rationale to believe that healthy persons are more robust than patients with LBP. In the most easy condition (standing on a stable support), people with LBP showed less sway than the healthy group. In contrast, in the most difficult condition (ballistic arm flexion on an unstable support), the healthy group showed less sway. These findings may indicate that the healthy group explores rather than controls, which in an easy condition results in more sway. However, in more complex conditions (having smaller safety margins), exploring may result in less sway, as seen in the most difficult trial. Despite, future research evaluating the COP in combination with a kinematical analysis of the body in postural control tasks may clarify this hypothesis. Beside linear outcome measures, also non-linear outcome measures are required to further evaluate this hypothesis (Mazaheri et al., 2013; Mazaheri et al, 20101; van Dieën et al. 2010).

Finally, it remains unclear if the proprioceptive steering of the subjects is caused by impaired signaling of the muscle spindles or by altered proprioceptive reweighting at brain level. Muscle spindle density may differ between subjects (Kokkorogiannis, 2004). In addition, individuals with LBP may have more fatigable back muscles and back muscle fatigue may compromise the multisegmental steering (Johanson et al., 2011; Mannion et al., 1997). In contrast, altered cerebrocortical activity is also shown during anticipatory postural adjustments in people with LBP (Jacobs et al., 2010). Brain imaging (e.g. fMRI) during postural control tasks while vibrating back and ankle muscles may further clarify the peripheral and/or central origin of proprioceptive changes in people with LBP.

3. Treatment and prevention of non-specific low back pain

Current project demonstrated that some proprioceptive postural control variables were associated with LBP and may play a causal role in the development of LBP. An important next step is to integrate the results of the current project in daily practice, both in the treatment of patients with LBP as in the development of prevention strategies.

Optimizing pelvic control may be fruitful based on the results in Study 2 and 3. The pelvis forms the basis of the bottom-up postural adjustment during sitting. This pelvic control must also be integrated in dynamic conditions such as moving from sitting to standing. Initiating the STSTS movement by a preparatory pelvic anterior rotation in people with a pelvic control impairment seems to be crucial to reduce the biomechanical load on the lumbosacral area (Cordo et al., 2003; Shum et al., 2009).

Optimizing the postural control strategy both in sitting and standing by stimulating persons to vary their postural strategies according to the postural condition (e.g. use of a more multi-segmental strategy during complex postural tasks) may be fruitful in the rehabilitation as well as in the prevention of LBP. This objective could be achieved be exercising multiple postural corrections at different levels during different functional positions (stable and unstable standing, stable and unstable sitting). Exercising in different postural positions may also enhance the variability in postural control strategies. This variability in motor task constituents reflects that people have several options to obtain a similar goal rather than relying on one single strategy (Harbourne and Stergiou, 2009). Lack of variability may lead to abnormal neural mapping and may disturb the motor function (Harbourne and Stergiou, 2009). Reduced variability in proprioceptive postural control strategies as demonstrated in people with LBP may result in impaired postural performance and may result in higher mechanical loads on the biomechanical system. During exercises patients must be challenged to explore postural control strategies rather than relying on one single rigid strategy as regularly done in ergonomic interventions and some core stability programs. During these interventions, people are often instructed to contract local stabilizing muscles (e.g. pelvic floor muscles, musculus transversus abdominis and musculi multifidi) in combination with diaphragmatic breathing. This co-contraction is exercised in different postural positions such as lying supine, sitting, standing, 4-point kneeling. Moreover, during dynamic activities as forward bending and STSTS, they are forced to keep this co-contraction to prevent spinal movements. The increased stiffness of this subsystem may not automatically lead to improvement of the performance of the total postural control system and in some conditions it degrades postural control (Reeves et al., 2006). Therefore, using the same strategy with increased stiffness in the lumbar area in all postural (static and dynamic) tasks may lead to decreased postural control of the body and may result in higher mechanical loads on the musculoskeletal system.

Focusing on the somatosensory perception during movements may be a crucial phase in the total rehabilitation program. Sensing, localizing and discriminating the incoming signals from the different regions of the body may facilitate the recovery of the proprioceptive function (Brumagne et al., 2013). For example, when performing different pelvic or lumbar movements (isolated lumbar or pelvic and combined), patients are instructed to feel when the movement starts, how it feels (sensing) and where the movements occur in this region (localizing). Subsequently, they are asked to differentiate which muscles are contracting or lengthening and in which direction the pelvis and the spine are moving (discriminating). This must be exercised in different static (e.g. standing, sitting) or dynamic (e.g. STSTS) postural positions dependent on the needs of the individual patient.

Kinesthetic imagery, the imagination of sensations elicited by movements, may help to reinforce the ability to choose the most optimal incoming proprioceptive signals according to the postural condition (Dickstein and Deutsch, 2007). Kinesthetic imagery is shown to modulate the somatosensory information, rather than visual imagery (Voisin et al., 2011). This may primarily occur at supraspinal level (Stinear et al., 2006). As a result kinesthetic imagery training may help to optimize the somatosensory reweighting in people with LBP. It may be followed by motor imagery training, imaging how a movement should be performed without any movement (Dickstein and Deutsch, 2007). Several studies already revealed the benefits of integrating motor imagery training in sports performance and balance control: it stimulates the cerebral activity during the performance of the motor task more than just performing the exercises (Giron et al., 2012; Rabahi et al., 2012; Rodrigues et al., 2010). Relating to people with LBP, imaging pelvic movement control in static postural conditions and pelvic movement initiation during dynamic tasks may optimize the variability in postural control strategies based on the results of current thesis. This type of exercises may optimize somatosensory reweighting and stimulate cortical activity (Guillot et al., 2009; Miller et al., 2010). In addition to motor control exercises, future research must clarify the usefulness of adding kinesthetic imagery and motor imagery training to the more conventional motor control exercises in people with LBP.

4. Conclusion

Current doctoral thesis showed a changed proprioceptive postural control in young people with mild LBP. They relied more on ankle muscle proprioceptive inputs compared to healthy controls and showed a reduced variability to choose the optimal postural control strategy according to the postural condition. In contrast to previous studies, the proprioceptive changes were specifically evaluated by means of muscle vibration. Moreover, the changes within the proprioceptive system were associated with delayed preparatory movement of the pelvis during the STSTS. Finally, the increased reliance on ankle proprioceptive signals could be identified as a risk factor for developing LBP. These results may have important consequences for the prevention and treatment of LBP.

5. References

1. Brumagne S, Cordo P, Verschueren S. Proprioceptive weighting changes in persons with low back pain and elderly persons during upright standing. Neurosci. Lett. 2004;366,(1):63-66.
2. Brumagne, S, Dolan P, and Pickar JG. Chapter 19: What is the relation between proprioception and low back pain? In: Hodges, PW, Cholewicki J., Van Dieën J. (Eds), Spinal Control: The Rehabilitation of Back Pain - State of the Art and Science. 2013;Elsevier Churchill Livingstone, Edinburgh, p. 219-230.ISBN:978-0-7020-4356-7.
3. Brumagne, S, Janssens L, Claeys K, and Pijnenburg M. Chapter 12: Altered variability in proprioceptive postural strategy in people with recurrent low back pain and healthy individuals. In:Hodges, PW, Cholewicki J, Van Dieën J (Eds),Spinal Control: The Rehabilitation of Back Pain - State of the Art and Science. 2013;Elsevier Churchil Livingstone, Edinburgh, p. 135-144.ISBN:978-0-7020-4356-7.
4. Brumagne S, Janssens L, Janssens E, Goddyn L. Altered postural control in anticipation of postural instability in persons with recurrent low back pain. Gait. Posture. 2008a;28,(4):657-662.
5. Brumagne S, Janssens L, Knapen S, Claeys K, Suuden-Johanson E. Persons with recurrent low back pain exhibit a rigid postural control strategy. Eur. Spine J. 2008b;17,(9):1177-1184.
6. Brumagne S, Lysens R, Spaepen A. Lumbosacral position sense during pelvic tilting in men and women without low back pain: test development and reliability assessment. J. Orthop. Sports Phys. Ther. 1999;29,(6):345-351.
7. Callaghan JP, and McGill SM. Intervertebral disc herniation: studies on a porcine model exposed to highly repetitive flexion/extension motion with compressive force. Clin. Biomech. 2001;16,(1):28-37.
8. Carragee EJ, Alamin TF, Miller JL, Carragee JM. Discographic, MRI and psychosocial determinants of low back pain disability and remission: a

prospective study in subjects with benign persistent back pain. Spine J. 2005;5,(1):24-35.

9. Cholewicki J, and McGill SM. Mechanical stability of the in vivo lumbar spine: implications for injury and chronic low back pain. Clin. Biomech. 1996;11,(1):1-15.
10. Cholewicki J, Silfies SP, Shah RA, Greene HS, Reeves NP, Alvi K, Goldberg B. Delayed trunk muscle reflex responses increase the risk of low back injuries. Spine 2005;30,(23):2614-2620.
11. Coin A et al. Trunk and lower limb fat mass evaluated by dual-energy X-ray absorptiometry in a 20- to 80-year-old healthy Italian population. Ann. Nutr. Metab 2012;61,(2):151-159.
12. Cordo PJ, Gurfinkel VS, Smith TC, Hodges PW, Verschueren SM, Brumagne S. The sit-up: complex kinematics and muscle activity in voluntary axial movement. J. Electromyogr. Kinesiol. 2003;13,(3):239-252.
13. Dankaerts W, O'Sullivan P, Burnett A, Straker L. Differences in sitting postures are associated with nonspecific chronic low back pain disorders when patients are subclassified. Spine 2006;31,(6):698-704.
14. della Volpe R, Popa T, Ginanneschi F, Spidalieri R, Mazzocchio R, Rossi A. Changes in coordination of postural control during dynamic stance in chronic low back pain patients. Gait. Posture. 2006;24(3):349-355.
15. Dickstein R, and Deutsch JE. Motor Imagery in Physical therapist Practice. Phys. Ther. 2007;87,942-953.
16. Gendreau M, Hufford MR, Stone AA. Measuring clinical pain in chronic widespread pain: selected methodological issues. Best Pract. Res. Cl. Rh. 2003;17(4):575-592.
17. George SZ, Fritz JM, Childs JD. Investigation of elevated fear-avoidance beliefs for patients with low back pain: a secondary analysis involving patients enrolled in physical therapy clinical trials. J. Orthop. Sports Phys. Ther. 2008;38,(2):50-58.
18. Giron EC, McIsaac T, Nilsen D. Effects of kinesthetic versus visual imagery practice on two technical dance movements: a pilot study. J. Dance. Med. Sci. 2012;16,(1):36-38.

19. Guclu DG, Guclu O, Ozaner A, Senormanci O, Konkan R. The relationship between disability, quality of life and fear-avoidance beliefs in patients with chronic low back pain. Turk. Neurosurg. 2012;22,(6):724-731.
20. Guillot A, Collet C, Nguyen VA, Malouin F, Richards C, Doyon J. Brain activity during visual versus kinesthetic imagery: an fMRI study. Hum. Brain Mapp. 2009;30,(7):2157-2172.
21. Harbourne RT, and Stergiou N. Movement variability and the use of nonlinear tools: principles to guide physical therapist practice. Phys. Ther. 2009;89,(3):267-282.
22. Heneweer H, Picavet HS, Staes F, Kiers H, Vanhees L. Physical fitness, rather than self-reported physical activities, is more strongly associated with low back pain: evidence from a working population. Eur. Spine J. 2012;21,(7):1265-1272.
23. Heneweer H, Vanhees L, Picavet HS. Physical activity and low back pain: a U-shaped relation? Pain 2009;143,(1-2):21-25.
24. Henry SM, Hitt JR, Jones SL, Bunn JY. Decreased limits of stability in response to postural perturbations in subjects with low back pain. Clin. Biomech. 2006;21,(9):881-892.
25. Jacobs JV, Henry SM, Nagle KJ. Low back pain associates with altered activity of the cerebral cortex prior to arm movements that require postural adjustment. Clin. Neurophysiol. 2010;121,(3):431-440.
26. Jamison RN, Raymond SA, Slawsby EA, McHugo GJ, Baird JC. Pain assessment in patients with low back pain: comparison of weekly recall and momentary electronic data. J. Pain. 2006;7(3):192-199.
27. Johanson E, Brumagne S, Janssens L, Pijnenburg M, Claeys K, Paasuke M. The effect of acute back muscle fatigue on postural control strategy in people with and without recurrent low back pain. Eur. Spine J. 2011;20(12):2152-9.
28. Kokkorogiannis T. Somatic and intramuscular distribution of muscle spindles and their relation to muscular angiotypes. J. Theor. Biol. 2004;229,(2):263-280.

29. Kuo YL, Tully EA, Galea MP. Video analysis of sagittal spinal posture in healthy young and older adults. J. Manipulative Physiol Ther. 2009;32,(3):210-215.
30. Kwok S, Canoy D, Soran H, Ashton DW, Lowe GD, Wood D, Humphries SE, Durrington PN. Body fat distribution in relation to smoking and exogenous hormones in British women. Clin. Endocrinol. 2011;doi: 10.1111/j.1365-2265.2011.04331.x.
31. Kwok S, Canoy D, Soran H, Ashton DW, Lowe GD, Wood D, Humphries SE, Durrington PN. Body fat distribution in relation to smoking and exogenous hormones in British women. Clin. Endocrinol. 2012;77,(6):828-833.
32. Leboeuf-Yde C, Nielsen J, Kyvik KO, Fejer R. Pain in the lumbar, thoracic or cervical regions: do age and gender matter? A population-based study of 34,902 Danish twins 20-71 years of age. BMC Musculoskelet. Disord. 2009;Doi: 10.1186/1471-2474-10-39.
33. Leinonen V, Kankaanpaa M, Luukkonen M, Kansanen M, Hanninen O, Airaksinen O, Taimela S. Lumbar paraspinal muscle function, perception of lumbar position, and postural control in disc herniation-related back pain. Spine 2003;28,(8):842-848.
34. MacDonald DA, Moseley GL, Hodges PW. The lumbar multifidus: Does the evidence support clinical beliefs? Man. Ther. 2006;11:254-263.
35. Mannion AF, Weber BR, Dvorak J, Grob D, Muntener M. Fibre type characteristics of the lumbar paraspinal muscles in normal healthy subjects and in patients with low back pain. J. Orthop. Res. 1997;15,(6):881-887.
36. Mazaheri M, Coenen P, Parnianpour M, Kiers H, van Dieën JH. Low back pain and postural sway during quiet standing with and without sensory manipulation: A systematic review. Gait Posture 2013;37(1):12-22.
37. Mazaheri M, Salavati M, Negahban H, Sanjari MA, Parnianpour M. Postural sway in low back pain: effects of dual tasks. Gait Posture 2010;31(1):116–121

38. McGill SM. Linking latest knowledge of injury mechanisms and spine function to the prevention of low back disorders. J. Electromyogr. Kinesiol. 2004;14,(1):43-47.
39. Miller KJ, Schalk G, Fetz EE, den NM, Ojemann JG, Rao RP. Cortical activity during motor execution, motor imagery, and imagery-based online feedback. Proc. Natl. Acad. Sci. U. S. A 2010;107,(9):4430-4435.
40. Mitchell T, O'sullivan PB, Burnett A, Straker L, Smith A, Thornton J, Rudd CJ. Identification of modifiable personal factors that predict new-onset low back pain: a prospective study of female nursing students. Clin. J. Pain 2010;26,(4):275-283.
41. Mitchell T, O'sullivan PB, Smith A, Burnett AF, Straker L, Thornton J, Rudd CJ. Biopsychosocial factors are associated with low back pain in female nursing students: a cross-sectional study. Int. J. Nurs. Stud. 2009;46,(5):678-688.
42. Mok NW, Brauer SG, Hodges PW. Hip strategy for balance control in quiet standing is reduced in people with low back pain. Spine 2004;29,(6):E107-E112.
43. Mok NW, Brauer SG, Hodges PW. Failure to use movement in postural strategies leads to increased spinal displacement in low back pain. Spine 2007;32,(19):E537-E543.
44. Mok NW, Brauer SG, Hodges PW. Changes in lumbar movement in people with low back pain are related to compromised balance. Spine 2011;36,(1):E45-E52.
45. Rabahi T, Fargier P, Rifai-Sarraj A, Clouzeau C, Massarelli R. Motor performance may be improved by kinesthetic imagery, specific action verb production, and mental calculation. Neuroreport 2012;23,(2):78-81.
46. Reeves NP, Narendra KS, Cholewicki J. Spine stability: the six blind men and the elephant. Clin. Biomech. 2007;22(3):266-274.
47. Rodrigues EC, Lemos T, Gouvea B, Volchan E, Imbiriba LA, Vargas CD. Kinesthetic motor imagery modulates body sway. Neuroscience 2010;169,(2):743-750.

48. Shum GL, Crosbie J, Lee RY. Three-dimensional kinetics of the lumbar spine and hips in low back pain patients during sit-to-stand and stand-to-sit. Spine 2007;32,(7):E211-E219.
49. Shum GL, Crosbie J, Lee RY. Energy transfer across the lumbosacral and lower-extremity joints in patients with low back pain during sit-to-stand. Arch. Phys. Med. Rehabil. 2009;90,(1):127-135.
50. Stinear CM, Byblow WD, Steyvers M, Levin O, Swinnen SP. Kinesthetic, but not visual, motor imagery modulates corticomotor excitability. Exp. Brain Res. 2006;168(1-2):157-164.
51. van Dieën J, Koppes L, Twisk J. Low back pain history and postural sway in unstable sitting, Spine2010;35(7):812–817.
52. Voisin JI, Mercier C, Jackson PL, Richards CL, Malouin F. Is somatosensory excitability more affected by the perspective or modality content of motor imagery? Neurosci. Lett. 2011;493,(1-2):33-37.
53. Yan S, Zhao X, Shen X, Yang L, Yuan X, Huang L, Zhang S. Abnormal Regional Body Fat Distribution Also Exists in Non-Obese Subjects with High Blood Pressure. Clin. Exp. Hypertens. 2013.
54. Zeidan MA, Lebron-Milad K, Thompson-Hollands J, IM JJ, Dougherty DD, Holt DJ, Orr SP, Milad MR. Test-retest Reliability During Fear Acquisition and Fear Extinction In Humans. CNS. Neurosc. Ther. 2012;18,(4):313-317.

Summary

Low back pain (LBP) and the reoccurrence of LBP is a major health problem in Western society with high social and economic consequences. Clinicians in daily practice are convinced that the group of patients with LBP is becoming still younger and the incidence of LBP in the late adolescence approximates the incidence of the adult population. Moreover, LBP at adolescent age is mostly idiopathic and may higher the risk for future episodes of LBP.

Idiopathic LBP or non-specific low back pain (LBP) is defined as pain in the lumbar and/or gluteal region without structural anatomical abnormalities as there are disc abnormalities, inflammation, fracture, tumor, etc. Pain may be caused or persist despite the absence of a nociceptive stimulus due to damage or injury. Altered proprioceptive postural control (postural changes, decreased postural robustness and changed proprioceptive afferent inputs) is frequently demonstrated as a contributing factor in LBP and may confirm that a real anatomical nociceptive stimulus is not always present in people with LBP.

A better understanding of the biological component of LBP in relation, and in addition to psychosocial factors, is important for a more rational approach to the management of LBP. Research into biological underlying causes and mechanisms may be of priority interest for the research in LBP for the next years. In addition to cross-sectional research, prospective studies are necessary to have more insight in the underlying causing mechanisms of LBP.

The general objective of this doctoral project is to generate a better insight in the proprioceptive postural control in young people with LBP versus healthy controls. Besides cross-sectional analysis also a prospective investigation was carried out to clarify a cause-effect relation. Four studies were performed to clarify this research question.

The variability in proprioceptive postural control strategies was investigated in **Chapter 2** (Study 1). Results showed a decreased variability in proprioceptive postural control strategies in young persons with LBP. Subjects with LBP showed a decreased capacity to switch to a more multisegmental strategy when this was more appropriate according to the postural condition (i.e., standing on an unstable support, sitting). Also a decreased postural robustness was shown in the more complex postural conditions. A reduced use of lumbar proprioceptive afference was associated with the decreased variability.

In **Chapter 3 (**Study 2), it was investigated if proprioceptive impairments demonstrated during static postural tasks in people with LBP are associated with an altered performance of a more dynamic task. As a dynamic task, the STSTS task was chosen. People with LBP and a concomitant decreased use of lumbar proprioceptive afference for standing postural control needed more time to perform five repetitions of the STSTS. The time differences were observed during the sit and stance phases (transition phases) and not during the focal movement phases. A decreased pelvic preparatory movement (slower onset of pelvic anterior rotation) was observed during the sit phases.

In C**hapter 4 (**Study 3), postural inter-correlations during usual standing and sitting in young healthy people were analyzed. In usual sitting, the pelvis was demonstrated to have most correlations with other spinal angles. In contrast, in usual standing the trunk angle showed most correlations with other spinal postural angles. However, correlations were mostly small to moderate, suggesting a between-subject variability in sagittal spinal posture without the existence of an optimal spinal posture.

Chapter five (Study 4) describes a prospective study that examined altered proprioceptive postural control, altered postural spinal angles and psychological variables as possible risk factors for future mild LBP within two years. Only an ankle-steered postural control strategy during stable standing was observed to be a risk factor for developing LBP in the near future. Other proprioceptive, postural, psychological or physical activity variables could not be identified as risk factors. It may be concluded that evaluating the proprioceptive postural control may help clinicians to identify people who are at risk for developing LBP.

This doctoral thesis brought more insight into the role of the proprioceptive system as underlying mechanism in LBP. A decreased capacity to upweight proprioceptive signals from the back muscles compared to the ankle muscles was demonstrated to be associated with LBP and to develop future LBP. Moreover, this impaired proprioceptive control also delayed the preparatory pelvic movement during the STSTS resulting in a

slower performance of this movement. These findings illustrate the importance of evaluating proprioception during postural control tasks rather than only focusing on the motor output when evaluating persons with LBP. Future research must clarify if the altered proprioceptive input is based on a decreased sensitivity of the muscle spindles and/or based on central changes.

Samenvatting

Lagerugpijn (LRP) en recidiverende opstoten van LRP zijn belangrijke gezondheidsproblemen in de Westerse maatschappij met aanzienlijke socio-economische consequenties. Therapeuten zijn overtuigd dat deze patiëntengroep steeds jonger wordt. De incidentie van LRP in de late adolescentie is bijna gelijk aan die van de volwassen populatie. Bovendien is LRP in de adolescentieperiode meestal idiopatisch. Opstoten van LRP op adolescentenleeftijd verhogen ook de kans op latere episodes van LRP.

Idiopathische LRP of a-specifieke lage rugpijn (A-LRP) wordt gedefiniëerd als pijn in de lumbale en/of gluteale regio zonder dat er structureel anatomische abnormaliteiten aanwezig zijn zoals letsels van de discus, inflammatie, fracturen, tumoren, etc. De pijn wordt dus veroorzaakt of in stand gehouden zonder duidelijk aanwijsbare nociceptieve prikkel op basis van anatomische schade of letsels. Gewijzigde proprioceptieve posturale controle (vb. gewijzigde houding, verminderde robuustheid van de houding en veranderde proprioceptieve signalen) is reeds in veel studies naarvoor gekomen als een belangrijke meespelende factor. Dit toont nogmaals aan dat een anatomische nociceptieve prikkel niet altijd aanwezig is bij mensen met A-LRP.

Om de behandeling van A-LRP nog beter te kunnen optimaliseren, is het noodzakelijk dat zowel de biologische als de psychosociale factoren nog beter begrepen worden. Onderzoek naar vooral de biologische onderliggende mechanismen zal het onderzoek in de eerstkomende jaren gaan domineren. Bovendien zijn naast cross-sectionele studies ook prospectieve studies noodzakelijk om nog meer inzicht te krijgen in die onderliggende, oorzakelijke mechanismen.

Het algemeen doel van dit doctoraatsonderzoek is nog meer inzicht te krijgen in de proprioceptieve posturale controle bij jongeren met A-LRP en gezonde leeftijdsgenoten. Naast een cross-sectioneel gedeelte is ook een prospectieve studie uitgevoerd om nog meer zicht te krijgen op de oorzaak/gevolg onduidelijkheid. Vier deelstudies zijn uitgevoerd om de onderzoeksvraag van dit project uit te klaren.

In **Hoofdstuk 2 (**Studie 1) werd de variabiliteit in proprioceptieve posturale controlestrategieën onderzocht. De resultaten van deze studie toonden een verminderde variabiliteit in proprioceptieve posturale controlestrategieën aan bij jongeren met A-LRP. De onderzochte mensen met A-LRP vertoonden een verminderde capaciteit om over te schakelen naar een meer multisegmenteel gestuurde houdingsstrategie wanneer dit het meest passend was voor die betreffende posturale conditie (staan op een onstabiele ondergrond, zitten). Bovendien werd ook een verminderde robuustheid van de posturale controle waargenomen in de meest complexe posturale taken. Een verminderd gebruik van proprioceptieve signalen uit de lage rug bleek geassocieerd te zijn met de verminderde variabiliteit.

In **Hoofdstuk 3** (Studie 2) werd onderzocht of de proprioceptieve veranderingen die waargenomen werden in stand bij mensen met A-LRP ook hun implicaties hebben op de uitvoering van meer dynamische taken. De zit-stand-zit beweging werd uitgekozen om dit te onderzoeken. Mensen met A-LRP, die dus minder proprioceptieve signalen vanuit de lage rug gebruiken, bleken meer tijd nodig te hebben om vijf maal de zit-stand-zit beweging uit te voeren. De tijdsverschillen bleken veroorzaakt te worden tijdens de zit- en standfasen (transitiefasen), maar niet tijdens de bewegingsfasen. Een vertraagde insturing van het bekken werd duidelijk waargenomen tijdens de zitfasen.

In Studie 3 **(Hoofdstuk 4)** werden correlaties tussen de verschillende wervelkolomregio's onderzocht tijdens de stand- en zithouding. Het bekken bleek de meeste correlaties te hebben met de andere posturale wervelkolomhoeken tijdens de gewoonte zithouding. Daartegenover vertoonde de romphoek meer correlaties met de andere posturale wervelkolomhoeken tijdens de gewoonte standhouding. Echter, de correlaties waren meestal slechs middelmatig. Dit suggereert een grote inter-subject variabiliteit in sagittale wervelkolompostuur, zonder dat er sprake kan zijn van één ideale sagittale houdingsopbouw ter hoogte van de wervelkolom.

Studie 4 (**Hoofdstuk 5)** beschrijft een prospectief onderzoek. Hierin werd onderzocht of gewijzigde proprioceptieve posturale controle, veranderde houding van de wervelkolom en psychosociale factoren als risicofactoren kunnen beschouwd worden om binnen de twee jaar milde A-LRP te ontwikkelen. Enkel een enkelgestuurde posturale controlestrategie tijdens het staan op stabiele ondergrond bleek een risicofactor te zijn om A-LRP te ontwikkelen in de nabije toekomst. Andere proprioceptieve variabelen, houding, psychologische factoren en fysieke activiteit konden niet als risicofactoren geïdentificeerd worden. Het evalueren van de sensorische input tijdens de proprioceptieve posturale controle kan voor clinici een hulpmiddel zijn om mensen te identificeren die een verhoogd risico lopen om A-LRP te ontwikkelen.

Dit doctoraatsproject bracht enkele extra inzichten in de rol van het proprioceptief systeem als onderliggend mechanisme voor het in stand houden van en/of het ontwikkelen van A-LRP. Een verminderd vermogen om de proprioceptieve prikkels van de lage rug meer te gebruiken t.o.v. die van de enkels bleek geassociëerd te zijn met huidige A-LRP en met het ontwikkelen van A-LRP in de nabije toekomst. Bovendien blijkt dit proprioceptief deficiet gepaard te gaan met een vertraagd insturen van het bekken tijdens de zit-stand-zit beweging, wat op zijn beurt leidt tot een vertraagd uitvoeren van deze beweging. Deze bevindingen tonen aan dat het evalueren van de proprioceptieve signalen tijdens posturale taken belangrijker is dan alleen te focussen op de motorische output van die taken tijdens het onderzoek van patiënten met A-LRP. Toekomstige wetenschappelijke studies moeten uitmaken of deze gewijzigde proprioceptieve sturing gebaseerd is op een verminderede sensitiviteit van de spierspoelen en/of veroorzaakt wordt door centrale veranderingen.

Bijstellingen

1. In een tijd waarin het budget van de ziekteverzekering steeds meer onder druk komt te staan, is het absoluut onverstandig om de terugbetaling van niet conventionele therapieën, waarvoor geen wetenschappelijke evidentie bestaat, in overweging te nemen.

2. In de medische wereld wordt er zowel bij zorgverstrekkers als patiënten nog te frequent curatief i.p.v. preventief gehandeld. Het is de taak van de overheid om hier nog meer aan sensibilisering te doen, maar ook (para)medici en patiënten dienen hun verantwoordelijkheid op te nemen.

3. De recente ontwikkelingen waarbij alle masteropleidingen aan de hogescholen inkantelen in de universiteit en waarbij de universiteit een uitgebreidere lokale aanwezigheid krijgt doorheen het Vlaamse land, kunnen voor perifere regio's extra kansen tot ontplooien geven. Laat ons niet uit het oog verliezen dat samenwerken op alle vlakken nog belangrijker zal worden om de onderzoeksoutput te optimaliseren.

About the author

Kurt Claeys was born on January 30, 1971 in Brugge, Belgium. He married Frances Van Loo in 2001. They have two children: Jarne and Myrthe.

In 1989 he graduated from high school at Sint-Lodewijkscollege in Brugge, Belgium. In 1993, he graduated as a Licentiaat in de Motorische Revalidatie en Kinesitherapie at the University of Gent, Belgium. After graduation, he started his own private practice for musculoskeletal physiotherapy in Zerkegem, Belgium. He completed specialization courses in the domain of the musculoskeletal physiotherapy such as Orthopedic Medicine Method Cyriax (1996) and Orthopedic Manual Therapy (2006). In 2001, he started as a lecturer at the Catholic University College (KHBO) in Brugge. In September 2007, he started his doctoral research project under supervision of Prof. Brumagne and Prof. Dankaerts. He continued to combine his research with teaching at the Department of Rehabilitation Sciences and Physiotherapy at the KHBO and with his activities in his private practice. Currently, he is appointed as chair of the Department of Rehabilitation Sciences and Physiotherapy at the new University Faculty of Brugge (Kulab).

Publications

1. Articles in internationally reviewed academic journals

- Janssens, L., Brumagne, S., McConnell, A., **Claeys, K.**, Pijnenburg, M., Burtin, C., Janssens, W., Decramer, M., Troosters, T. (2013). Proprioceptive changes impair balance control in individuals with chronic obstructive pulmonary disease. *PLoS One*, 8 (3), e57949.
- Janssens, L., Troosters, T., McConnell, A., Pijnenburg, M., **Claeys, K.**, Brumagne, S. (2012). Postural strategy and back muscle oxygenation during inspiratory muscle loading. *Medicine and Science in Sports and Exercise*, 45(7), 1355-1362.
- **Claeys, K.**, Dankaerts, W., Janssens, L., Brumagne, S. (2012). Altered preparatory pelvic control during the sit-to-stance-to-sit movement in people with nonspecific. *Journal of Electromyography and Kinesiology*, *22* (6), 821-828.
- Johanson, E., Brumagne, S., Janssens, L., Pijnenburg, M., **Claeys, K.**, Paasuke, M. (2011). The effect of acute back muscles fatigue on postural control in people with and without recurrent low back pain. *European Spine Journal*, *20* (12), 2152-2159.
- **Claeys, K.**, Brumagne, S., Dankaerts, W., Kiers, H., Janssens, L. (2011). Decreased variability in postural strategies in young people with non-specific low back pain is associated with altered proprioceptive reweighting. *European Journal of Applied Physiology*, 111(1), 115-123.
- Brumagne, S., Janssens, L., Knapen, S., **Claeys, K.**, Süüden-Johanson, E. (2008). Persons with recurrent low back pain exhibit a rigid postural control strategy. European Spine Journal 17 (9), 1177-1184.

2. Articles in other academic journals

- **Claeys, K.**, Janssens, L., Brumagne, S. (2011). Proprioceptieve posturale controle bij personen met aspecifieke lagerugpijn. *Neuron, 16* (3), 88-93.
- **Claeys, K.**, Janssens, L., Brumagne, S. (2010). Proprioceptieve posturale controle bij personen met aspecifieke lagerugpijn. *Ortho-Rheumato,* 8(6), 235-240.

3. Articles in academic book, internationally recognized scientific publisher

- Brumagne, S., Janssens, L., **Claeys, K.**, Pijnenburg, M. (2013). Altered variability in proprioceptive postural strategy in people with recurrent low back pain. In: Hodges P., Cholewicki J., Van Dieen J. (Eds.), *Spinal Control: The Rehabilitation of Back Pain - State of the art and Science*, Chapt. 12. Edinburgh: Elsevier Churchill Livingstone, 135-144.

4. Meeting abstracts, presented at international scientific conferences and symposia, published or not published in proceedings or journals

- Brumagne, S., Janssens, L., Pijnenburg, M., **Claeys, K.** (2012). Processing of conflicting proprioceptive signals during standing in people with and without recurrent low back pain. XIX Biennial Conference of the International Society of Electrophysiology and Kinesiology. Brisbane, 19-21 July 2012.
- **Claeys, K.**, Brumagne, S., Dankaerts, W., Janssens, L. (2011). Decreased variability in postural strategy in people with non-specific low back pain during standing and sitting. *Physiotherapy: vol. 97* (S1). WCPT. Amsterdam, 20-23 June 2011, Abstract No. RR-PL-2827.

- **Claeys, K.**, Brumagne, S., Dankaerts, W., Janssens, L. (2010). Decreased variability in postural strategy in people with non-specific low back pain during standing and sitting. *Balanced Solutions: Effective implementation of evidence based research*. Interdisciplinary World Congress on Low Back and Pelvic Pain, Los Angeles. Los Angeles, 2010, 587-588.
- **Claeys, K.**, Brumagne, S., Dankaerts, W. (2010). Decreased use of lumbar proprioceptive inputs reduces variability in postural strategy in people with non-specific low back pain. *European Spine Journal: vol. 19* (Suppl 3). Eurospine Annual Meeting. Vienna, 15-17 September 2010, S284-S285.
- **Claeys, K.**, Brumagne, S., Dankaerts, W., Janssens, L., Kiers, H. (2009). Differences in proprioceptive postural control during the sit-to-stand-to-sit movement between persons with non-specific low back pain and healthy controls on an unstable support surface. ISPGR International Conference. Bologna, 21-25 June 2009.
- **Claeys, K.**, Brumagne, S., Dankaerts, W., Kiers, H., Janssens, L. (2009). Differences in proprioceptive postural control during the sit-to-stand-to-sit movement between persons with non-specific low back pain and healthy controls on a stable surface. ISPGR International Conference. Bologna, 21-25 June 2009.
- Janssens, L., Brumagne, S., Polspoel, K., **Claeys, K.**, McConnell, A. (2009). Altered proprioceptive control induced by inspiratory muscles fatigue in persons with and without recurrent low back pain. ISPGR International Conference. Bologna, 21-25 June 2009.
- Janssens, L., Brumagne, S., Polspoel, K., **Claeys, K.**, Troosters, T., McConnell, A. (2009). Acute inspiratory muscles fatigue induces a rigid proprioceptive postural control strategy in persons with and without low back pain. Annual Meeting (36th) of the International Society for the Study of the Lumbar Spine. Miami, 4-8 May 2009.

5. Science popularization

- **Claeys, K.** (2013). Proprioceptive postural control in people with non-specific low back pain: an update. Gastcollege voor Physiotherapeuten. Hanzehogeschool Groningen, 27 May 2013.
- **Claeys, K.** (2013). The proprioceptive system: evaluation and treatment. Lezing voor studenten physiotherapie uit Groningen. KHBO Brugge, 07 February 2013.

6. Collaboration on Master theses

- Grosemans, E., Brumagne, S., **Claeys, K**. (2012). Decreased variability in postural control strategies: a risk factor for low back pain, *non-published master thesis*, KU Leuven, 37 p.
- Kerstens, N., Dankaerts, W., **Claeys, K.** (2012). Houdingsanalyse van de wervelkolom tijdens tillen bij jongeren met en zonder lage rugpijn, *non-published master thesis*, KU Leuven, 40 p.
- Vandeputte, S., Dankaerts, W., **Claeys, K.** (2012). Postural differences and inter-correlations during standing and lifting in a population with non-specific low back pain and healthy controls, *non-published master thesis*, KU Leuven, 40 p.
- Van Le, T., Dankaerts, W., **Claeys, K.** (2012). Altered thoracic sagittal spinal alignment in patients with neck pain, *non-published master thesis*, KU Leuven, 37 p.
- Dierick, J., Dankaerts, W., **Claeys, K.** (2011). Relationship between psychosocial factors, spinal postures and spinal pain in a young population – a prospective study, *non-published master thesis*, KU Leuven, 32 p.
- Gevers, S., Dankaerts, W., **Claeys, K.** (2011). Prospective evaluation of spinal posture in a young student population and the influence of spinal pain, *non-published master thesis*, KU Leuven, 29 p.
- Mahmood, A., Brumagne, S., **Claeys, K.** (2011). Proprioceptive postural control during the sit-to-stand-to-sit movement on an unstable surface in healthy people and

people with non-specific low back pain, *non-published master thesis*, KU Leuven, 31 p.

- Vanderheyden, Y., Brumagne, S., **Claeys, K.** (2011). Proprioceptive postural control during upright standing in people with and without low back pain: a prospective study, *non-published master thesis*, KU Leuven, 32 p.
- Crol, A., Brumagne S., **Claeys, K.** (2010). Postural control during the sit-to-stance-to-sit movement in individuals with and without low back pain, *non-published master thesis*, KU Leuven, 29 p.
- De Boom, L., Hex, L., Dankaerts, W., **Claeys, K**. (2010). Relationship between spinal postures and psychosocial factors and the influence of low back pain in a young student population - a cross-sectional study, *non-published master thesis*, KU Leuven, 31 p.
- Raftopoulou, A., Claeys, M., Dankaerts, W., **Claeys, K.** (2010). Correlations between cervical, thoracic and lumbar posture in sitting and its relationship with low back pain, *non-published master thesis*, KU Leuven, 27 p.
- Tilgner, N., Brumagne, S., **Claeys, K.** (2010). Proprioceptive postural control in patients with low back pain and healthy controls during standing and sitting on a stable support surface, *non-published master thesis*, KU Leuven, 28 p.
- Vermeir, P., Brumagne S., **Claeys, K.** (2010). Proprioceptive postural control during upright standing in young individuals with and without non-specific low back pain, *non-published master thesis,* KU Leuven, 33 p.

Printed by PROCOPIA NV
Ambachtenlaan 29
3001 Heverlee
Belgium
0032 (16)38.36.34
info@procopia.be
www.procopia.be